July 1990
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RESPIRATORY CARE

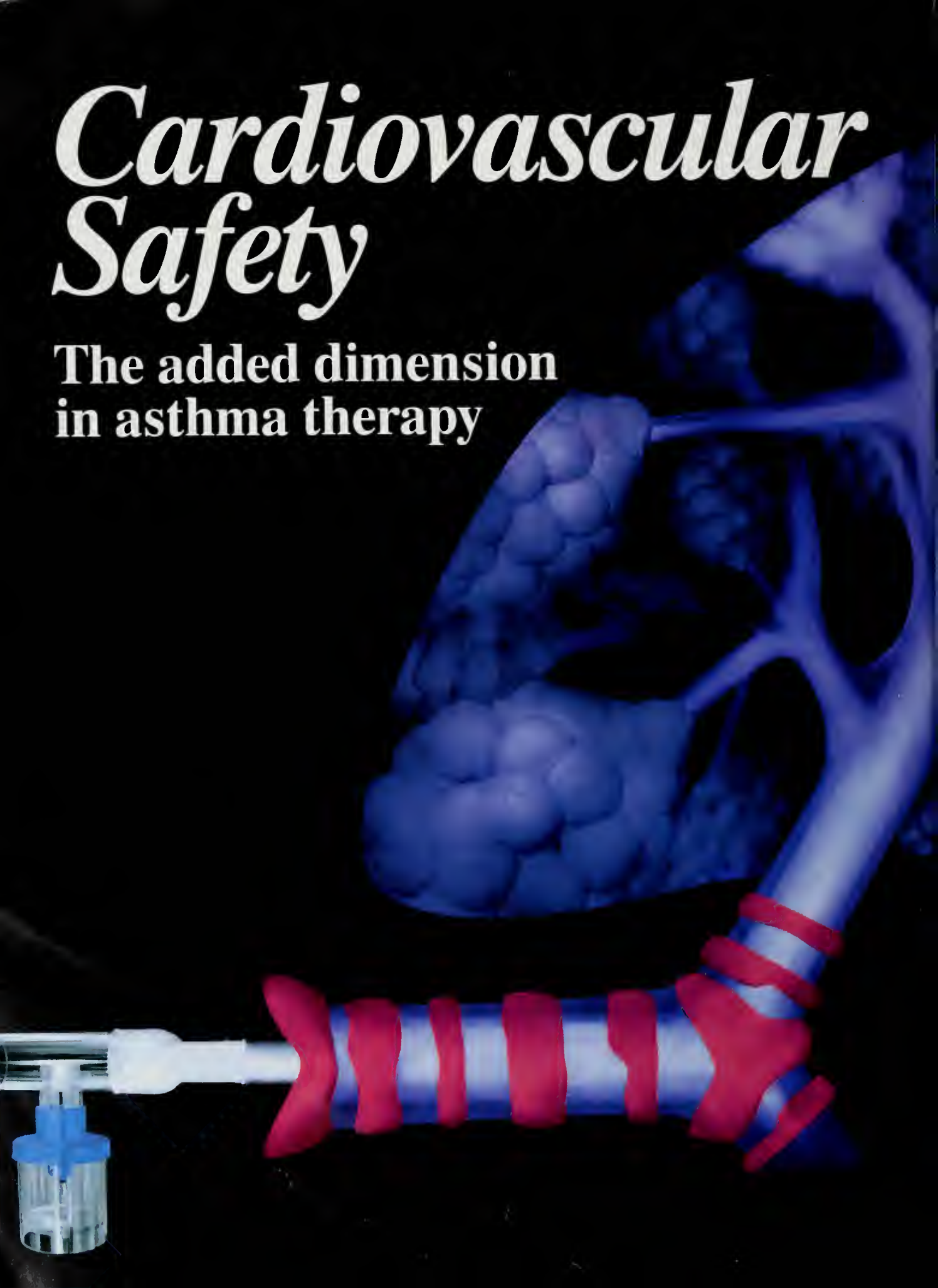
A MONTHLY SCIENCE JOURNAL
35TH YEAR—ESTABLISHED 1956

SPECIAL ISSUE
NONINVASIVE MONITORING
PART II

- Measurement of Energy Expenditure: Instrumentation, Practical Considerations, and Clinical Application
- Measuring and Monitoring Lung Volumes Outside the Laboratory
- Lung Mechanics Determinations at the Bedside: Instrumentation and Clinical Application
- Ventilatory Pattern Monitoring
- Noninvasive Hemodynamic Monitoring
- Monitoring of Respiratory Function Outside the Hospital
- Resource Management
- 1989 Journal Conference Summary

Cardiovascular Safety

The added dimension in asthma therapy



- **Less than 1% incidence of tachycardia in patients using Alupent Inhalation Aerosol[1]**
- **No adverse cardiovascular effects noted in two clinical comparisons of Alupent and albuterol Inhalation Solutions[2,3]**

Adverse reactions similar to those of other sympathomimetic agents*

*As with other sympathomimetic agents, Alupent should be used with caution in patients with cardiovascular disorders, including ischemic heart disease, hypertension or cardiac arrhythmias, in patients with hyperthyroidism or diabetes mellitus, and in patients unusually sensitive to sympathomimetic amines or who have convulsive disorders. Significant changes in systolic and diastolic blood pressure could be expected to occur in some patients after use of any beta-adrenergic bronchodilator.

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Alupent
(metaproterenol sulfate)

Inhalation Aerosol
10 mL 15 mg/mL (each metered dose delivers 0.65 mg metaproterenol sulfate)

Inhalation Solution
5% in 10 mL and 30 mL bottles; unit dose 0.6% and 0.4%

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Please see following page for brief summary of Prescribing Information.

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*10 mL; 15 mg per mL (each metered dose delivers 0.65 mg metaproterenol sulfate)

Bronchodilator

Brief Summary of Prescribing Information

CONTRAINDICATIONS Use in patients with cardiac arrhythmias associated with tachycardia is contraindicated

Although rare, immediate hypersensitivity reactions can occur. Therefore, Alupent® (metaproterenol sulfate USP) is contraindicated in patients with a history of hypersensitivity to any of its components.

WARNINGS Excessive use of adrenergic aerosols is potentially dangerous. Fatalities have been reported following excessive use of Alupent® (metaproterenol sulfate USP) as with other sympathomimetic inhalation preparations, and the exact cause is unknown. Cardiac arrest was noted in several cases.

Alupent, like other beta adrenergic agonists, can produce a significant cardiovascular effect in some patients, as measured by pulse rate, blood pressure, symptoms, and/or ECG changes. As with other beta adrenergic aerosols, Alupent can produce paradoxical bronchospasm (which can be life-threatening). If it occurs, the preparation should be discontinued immediately and alternative therapy instituted.

Alupent should not be used more often than prescribed. Patients should be advised to contact their physician in the event that they do not respond to their usual dose of a sympathomimetic amine aerosol.

PRECAUTIONS General Extreme care must be exercised with respect to the administration of additional sympathomimetic agents.

Since metaproterenol is a sympathomimetic amine, it should be used with caution in patients with cardiovascular disorders, including ischemic heart disease, hypertension or cardiac arrhythmias, in patients with hyperthyroidism or diabetes mellitus, and in patients who are unusually responsive to sympathomimetic amines or who have convulsive disorders. Significant changes in systolic and diastolic blood pressure could be expected to occur in some patients after use of any beta adrenergic bronchodilator

Information for Patients Extreme care must be exercised with respect to the administration of additional sympathomimetic agents. A sufficient interval of time should elapse prior to administration of another sympathomimetic agent

Drug Interactions Other beta adrenergic aerosol bronchodilators should not be used concomitantly with Alupent® (metaproterenol sulfate USP) because they may have additive effects. Beta adrenergic agonists should be administered with caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants, since the action of beta adrenergic agonists on the vascular system may be potentiated.

Carcinogenesis/Mutagenesis/Impairment of Fertility In an 18-month study in mice, Alupent produced an increase in benign ovarian tumors in females at doses corresponding to 320 and 640 times the maximum recommended dose (based on a 50 kg individual). In a 2-year study in rats, a nonsignificant incidence of benign leiomyomata of the mesovarium was noted at 640 times the maximum recommended dose. The relevance of these findings to man is not known. Mutagenic studies with Alupent have not been conducted. Reproduction studies in rats revealed no evidence of impaired fertility.

Pregnancy/Teratogenic Effects *PREGNANCY CATEGORY C* Alupent has been shown to be teratogenic and embryotoxic in rabbits when given orally in doses 620 times the human inhalation dose and 62 times the human oral dose. There are no adequate and well-controlled studies in pregnant women. Alupent should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Oral reproduction studies in mice, rats and rabbits showed no teratogenic or embryocidal effects at 50 mg/kg corresponding to 310 times the human inhalation dose and 31 times the human oral dose. Teratogenic effects in the rabbit included skeletal abnormalities and hydrocephalus with bone separation.

Nursing Mothers It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Alupent is administered to a nursing woman.

Pediatric Use Consult package insert for age limit.

ADVERSE REACTIONS Adverse reactions are similar to those noted with other sympathomimetic agents. Adverse reactions such as tachycardia, hypertension, palpitations, nervousness, tremor, nausea and vomiting have been reported.

The most frequent adverse reaction to Alupent® (metaproterenol sulfate USP) administered by metered-dose inhaler among 251 patients in 90-day controlled clinical trials was nervousness. This was reported in 6.8% of patients. Less frequent adverse experiences, occurring in 1% to 4% of patients, were headache, dizziness, palpitations, gastrointestinal distress, tremor, throat irritation, nausea, vomiting, cough and asthma exacerbation. Tachycardia occurred in less than 1% of patients.

HOW SUPPLIED Inhalation Aerosol Each Alupent® (metaproterenol sulfate USP) Inhalation Aerosol contains 150 mg of metaproterenol sulfate as a micronized powder in inert propellants. Each metered dose delivers through the mouthpiece 0.65 mg metaproterenol sulfate (each mL contains 15 mg). Alupent Inhalation Aerosol with Mouthpiece, net contents 14 g (10 mL), equipped with blue protective cap. Alupent Inhalation Aerosol Refill, net contents 14 g (10 mL).

Inhalation Solution Alupent Inhalation Solution is supplied as a 5% solution in bottles of 10 mL or 30 mL with accompanying calibrated dropper.
Alupent Inhalation Solution Unit-dose Vial is supplied as a 0.4% or 0.6% clear colorless or nearly colorless solution containing 2.5 mL, with 25 vials per box.

Syrup Alupent is available as a cherry-flavored syrup, 10 mg per teaspoonful (5 mL), in 16 fl. oz. bottles.

Tablets Alupent is supplied in 2 dosage strengths as scored, round white tablets in bottles of 100. Tablets of 10 mg coded BI/74. Tablets of 20 mg coded BI/72.

Consult package insert before prescribing. AL-BS-10/89

References:

1. Alupent® (metaproterenol sulfate) Inhalation Aerosol full Prescribing Information
2. Krainson JP. Metaproterenol and albuterol nebulizer solutions--a double-blind crossover study. Presented at the 46th Annual Meeting of the American College of Allergy and Immunology, November 13, 1989, Orlando, Fla.
3. Habib MP, Campbell SC, Shon BY, Pinnas JL. A comparison of albuterol and metaproterenol nebulizer solutions. *Ann Allergy*. 1987;58:421-424

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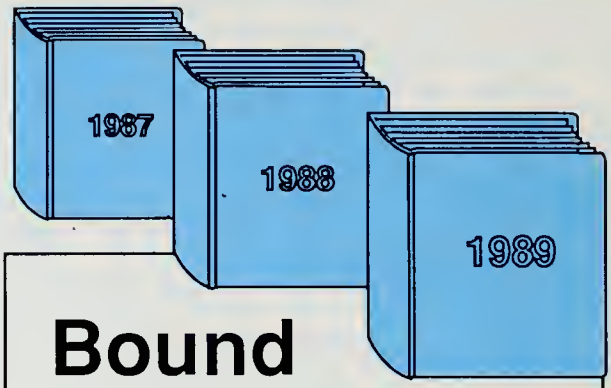

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Chairmen and Guest Editors

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Applegate WB, Blass JP, Williams TF. Instruments for the functional assessment of older patients (review). N Engl J Med 1990;322:1207-1214.

Douglas NJ, Flenley DC. Breathing during sleep in patients with obstructive lung disease (review). Am Rev Respir Dis 1990;141:1055-1070.

Sibille Y, Reynolds HY. Macrophages and polymorphonuclear neutrophils in lung defense and injury (review). Am Rev Respir Dis 1990;141:471-501.

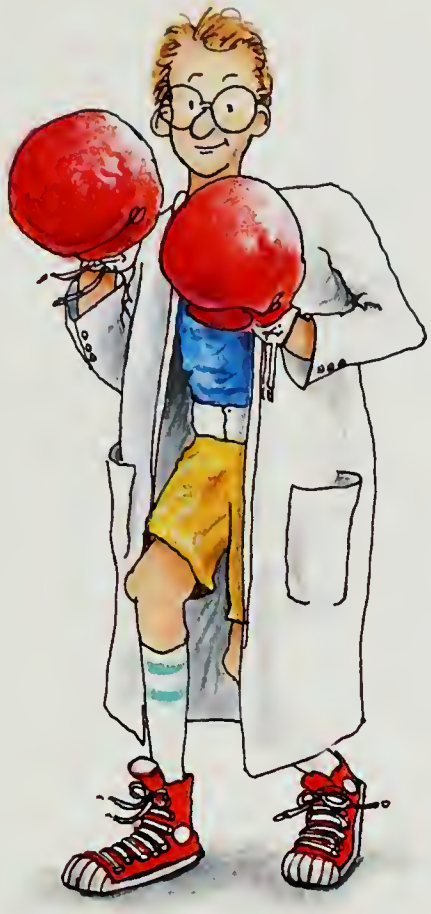
American College of Chest Physicians/Society of Critical Care Medicine Consensus Panel. Ethical and moral guidelines for the initiation, continuation, and withdrawal of intensive care. Chest 1990;97:949-961.

How Many Patients Are Necessary to Assess Test Performance—CF Arkin, MS Wachtel. JAMA 1990; 263:275.

Test performance characteristics are important in assessing the clinical usefulness of laboratory tests and serve as a basis for comparing one test to another. Statistical comparisons of performance characteristics are meaningful only when they can detect medically important differences; that is, when they provide adequate statistical power. This requires choosing the appropriate sample size in determining the performance characteristics of interest. Using standard formulas, we designed tables that provide such sample size requirements. Example problems of sample size determination in laboratory test comparisons are given. Used appropriately, this approach should result in better studies of laboratory tests and fewer meaningless negative studies.

Ventilatory Muscle Loads and the Frequency-Tidal Volume Pattern during Inspiratory Pressure-Assisted (Pressure-Supported) Ventilation—NR MacIntyre, NE Leatherman. Am Rev Respir Dis 1990;141:327.

Pressure support ventilation (PSV) is a new form of mechanical ventilatory support that assists a patient's spontaneous ventilatory effort with a clinician-selected amount of inspiratory pressure. In order to assess the muscle unloading effect and the ventilatory pattern response to increasing levels of this inspiratory pressure assist, we first utilized a computer respiratory system model with variable alveolar ventilation demands and impedances. From this model, we calculated ventilatory muscle loads (expressed either as the work/min or as the pressure time index) during simulated, unassisted breathing and during simulated breathing with levels of inspiratory pressure assist up to that which resulted in a V_T of 800 mL and no work being performed by the muscles (defined as PSV_{max} for the model conditions being studied). The optimal ventilatory pattern (ie, frequency-tidal volume) under each ventilation and impedance condition was defined as that which resulted in minimal muscle load. Under these model conditions, we found that PSV_{max} ranged from 5 to 41 cm H_2O and that as the level of inspiratory pressure assist was increased from zero to PSV_{max}, there was a biphasic response of both the ventilatory muscle loading and the ventilatory pattern. Specifically, at low levels of inspiratory pressure assist, the model predicted that the applied pressure would only partially unload the ventilatory muscles. Continued muscle energy expenditure would thus still be required, whereas the ventilatory pattern would change little. Conversely, at higher levels of inspiratory pressure



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assist, the model predicted that the applied pressure would be sufficient to completely unload the ventilatory muscles. Under these conditions, no muscle energy would be required, and further increases in inspiratory pressure assist would result primarily in larger V_T (and consequently smaller frequency). The crossover from partially loaded to unloaded conditions was found to be variable (ie, 30 to 90% of PSV_{max}) and depended upon both the ventilation and the impedance conditions studied. We then reanalyzed, within the context of this model, data from a previous study of 15 patients receiving various levels of inspiratory pressure assist sufficient to prevent clinical evidence of ventilatory muscle fatigue. From this analysis, both predicted and observed estimates of load at PSV_{max} were near zero. Moreover, in accordance with model predictions, the observed ventilatory pattern appeared biphasic, with the crossover between partially loaded and unloaded conditions occurring at 82 to 90% of PSV_{max}.

The Presence of Airway Reactivity before the Development of Asthma—RJ Hopp, RG Townley, RE Biven, AK Bewtra, NM Nair. Am Rev Respir Dis 1990;141:2.

Exaggerated airway reactivity is an essential component of the current asthmatic. It is not clear, however, if airway reactivity is genetically determined or acquired. To examine the possibility that increaed bronchial reactivity exists prior to the development of asthma, we report on 20 subjects who were studied before and after the onset of clinical asthma. Subjects were part of a larger ongoing study of the Natural History of Asthma. Thirteen subjects indicated by their answers to the National Heart, Lung, and Blood Institute respiratory questionnaire that they were not asthmatic at their initial visit. Seven subjects had pulmonary symptoms on their initial visit but had not been diagnosed as asthmatic. Bronchial reactivity was assessed using a standardized methacholine challenge. For the 20 subjects, there was a mean interval of 3.5 yr between the initial visit and the diagnosis of asthma. Ten of 13 nonasthmatic subjects had moderate or strongly positive responses (208 breath units or less) to methacholine prior to onset of asthma. These 13 subjects were compared to age- and sex-matched controls, from both asthmatic and nonasthmatic families who had not become asthmatic. There was a difference in bronchial responses at the initial visit between the 13 study subjects and their control subjects from nonasthmatic families, but not between the subjects and their control from asthmatic families. Five of 7 subjects with pulmonary symptoms had responses of 100 breath units or less. Overall, 19 of 20 subjects had strongly positive responses to methacholine after the diagnosis of asthma was established. The results show that enhanced airway reactivity usually precedes the development of asthma, which could support a genetic basis for it.

Respiratory and Skeletal Muscle Function in Steroid-Dependent Bronchial Asthma—C Picado, JA Fiz, JM Montserrat, et al. Am Rev Respir Dis 1990;141:14.

Respiratory and skeletal (deltoid) muscle strength were evaluated in 34 oral steroid-dependent asthmatics by use of maximal inspiratory and expiratory pressures and a myometer. The patients were compared to age- and sex-matched asthmatics who had never been on continuous oral steroid treatment. Endurance time was also studied in ten steroid-dependent asthmatics and ten controls using a pressure threshold breathing device. Nutritional status was assessed from body weight, midarm circumference, triceps skinfold (TSF), prealbumin, albumin, and total protein. An open biopsy from deltoid muscle was taken from nine steroid-dependent asthmatics and the diameter of type 1 and type 2 fibers was measured by a morphometric study. No differences were found between study and control groups either in respiratory and skeletal muscle strength or in endurance time. Steroid-dependent asthmatics showed a decrease in TSF, total protein, albumin, and potassium serum levels when compared with the control group but differences were not statistically significant after Bonferroni's adjustment for multiple comparison studies. Transversal diameter of type 2 fibers was significantly correlated with the percentage of ideal weight ($r = 0.75$, $p < 0.05$), but not with average daily dose of steroids nor with the length of steroid treatment. Our results support the clinical impression that steroids at the doses usually administered in chronic severe asthma do not cause muscular weakness. We also found that malnutrition rather than corticosteroids is the most important contributory factor to type 2 muscle fiber atrophy in steroid-dependent asthma.

Pulsed Nasal and Transtracheal Oxygen Delivery—BL Tiep, KL Christopher, BT Spofford, JR Goodman, PD Worley, SL Macy. Chest 1990;97:364.

Oxygen conserving devices, the transtracheal oxygen (TTO) catheters, electronic pulsed demand oxygen delivery systems (DODS), and reservoir cannulas, increase efficacy of oxygen delivery; TTO also improves cosmetic appearance, comfort and compliance. We speculated that pulsing of oxygen transtracheally can increase efficiency of TTO. We modified the DODS to include settable delays and a rapid pre-inspiratory trigger. The first part of the study was

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performed with nasal oxygen on 7 subjects and the second part with TTO on 17 subjects. Nasal oxygen results indicate improved delivery efficiency with more rapid response. The TTO results indicate no significant change for each delay setting, but there was improvement in delivery efficiency when DODS was combined with TTO vs continuous flow TTO. Thus, early inspiratory delivery increases efficiency of oxygen therapy. Small delays in response time are critical in nasal delivery but not important in TTO. Pulsed TTO is more efficient than continuous-flow TTO and merits long-term studies.

Comparison of Flow-Resistive Work Load due to Humidifying Devices—M Nishimura, MK Nishijima, T Okada, N Taenaka, I Yoshiya. Chest 1990;97:600.

There are many kinds of humidifying devices. We evaluated six humidifiers from the viewpoint of added work load (AWL). The AWL was obtained by calculating the area difference between pressure-volume tracings obtained without and with humidifiers. We examined the effect of pressure monitoring sites on AWL when a humidifier is placed, we measured AWL at three different pressure monitoring sites. The AWL was affected significantly by the pressure monitoring site for the ventilator. When a pressure monitor sensor was placed on the inspiratory limb between the inspiratory valve and humidifiers, the ventilator was not able to compensate for the pressure drop caused by impedance characteristics of the humidifier equipment. This resulted in significant inspiratory AWL on the patient. Thus, humidifying devices should be carefully selected from the viewpoint of not only humidifying capability but also AWL. Furthermore, we must recognize the importance of the pressure monitoring site for the ventilator.

A Comparison of Seven-Point and Visual Analogue Scales: Data from a Randomized Trial—R Jaeschke, J Singer, GH Guyatt. Controlled Clinical Trials 1990;11:43.

Many controlled trials rely on subjective measures of symptoms or quality of life as primary outcomes. The relative merits of different response options for these instruments is an important issue. Therefore, we compared the responsiveness and validity of 7-point versus visual analogue scales (VAS) in a questionnaire measuring quality of life in chronic heart failure in a double-masked crossover trial of digoxin versus placebo. The VAS and 7-point scale versions of the questionnaire were administered to 20 patients during 7 weeks on digoxin and 7 weeks on placebo. The order of administration of the questionnaires was determined by random allocation. The ability of the two methods to distinguish digoxin and placebo, and their correlation with other measures, were comparable. We conclude that the two methods of presenting response options show comparable responsiveness and validity in a randomized trial setting. The ease of administration and interpretation of the 7-point scale recommend its use.

Emphysematous Change in Chronic Asthma in Relation to Cigarette Smoking: Assessment by Computed Tomography—Y Kondoh, G Taniguchi, S Yokoyama, F Taki, K Takagi, and T Satake. Chest 1990; 97:845.

To evaluate the occurrence and the degree of emphysema in chronic asthma in relation to the effect of cigarette smoking, we examined 35

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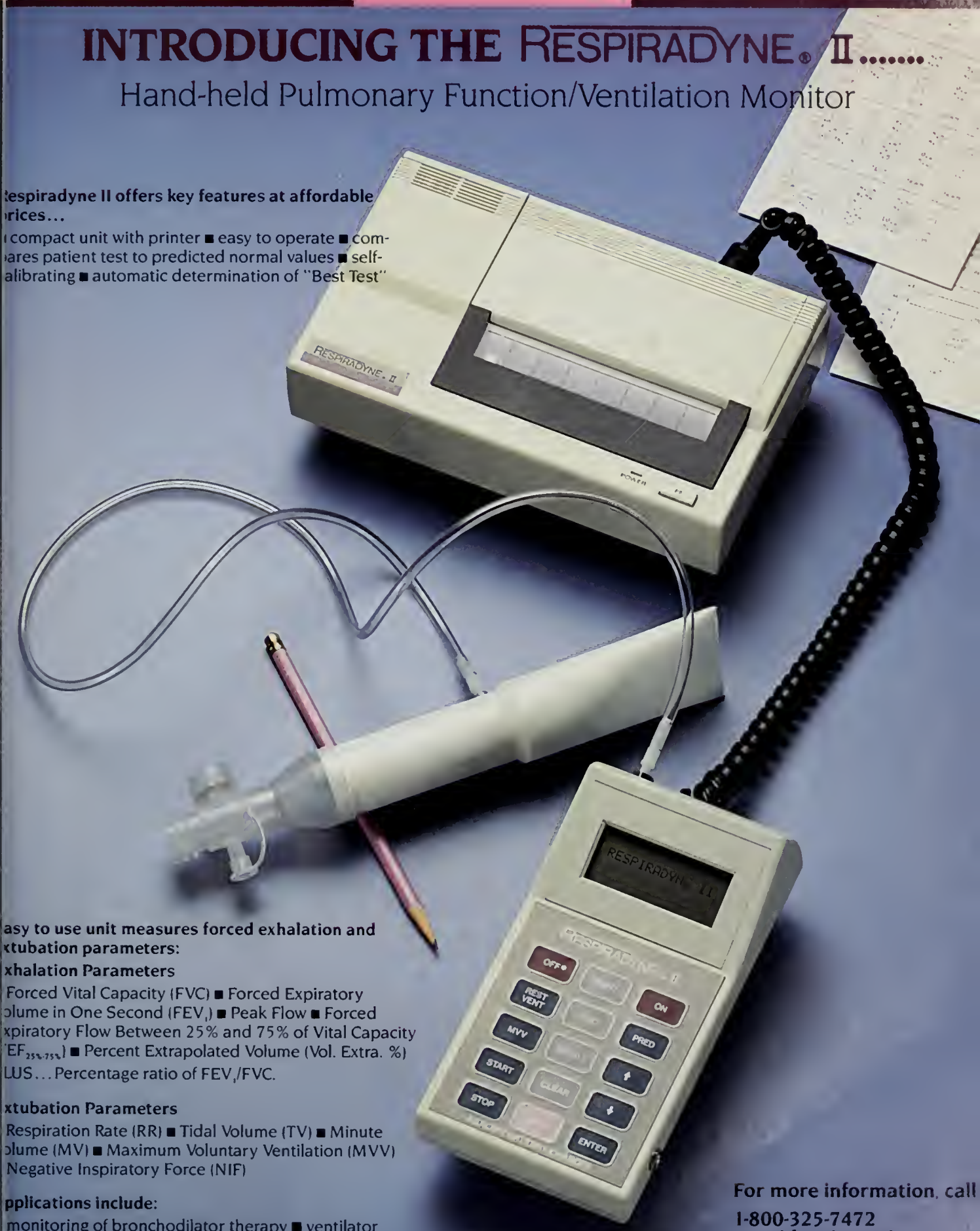

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subjects with irreversible airway obstruction (17 nonsmokers and 18 smokers). We performed pulmonary function testing and computed tomography (CT) scans on all subjects. The emphysema score (ES) was assessed by a visual scoring system on CT scans. Between nonsmokers and smokers, there was a significant difference in the ES ($p < 0.05$), but not in the FEV_1, TLC, and D_{sb}/V_A (expressed as percent predicted values). The ES was 2.3 ± 4.7% (mean SD) in nonsmoking subjects and 13.7 ± 16.7% in smoking subjects. In all subjects the ES showed significant correlations with D_{sb}/V_A ($p < 0.001$) and pack-years of cigarette consumption ($p < 0.001$) but did not show correlations with FEV_1 and with TLC. We concluded that emphysema can occur in smoking asthmatic subjects because of the effect of cigarette smoking, and CT scans are useful for detecting this emphysematous change.

Single-Breath Carbon Monoxide Diffusing Capacity Prediction Equations from a Mediterranean Population—J Roca, R Rodriguez-Roisin, E Cobo, F Burgos, J Perez, JL Clausen. Am Rev Respir Dis 1990;141:1026.

Because of unanswered questions about prediction equations for the single-breath carbon monoxide diffusing capacity (D_{LCO}) and as a part of a larger collaborative project, standardized D_{LCO} measurements were carried out in a selected sample of 361 healthy nonsmoking volunteers (194 men and 167 women) living in the Barcelona metropolitan area (Spain). Except for the test F_{IO_2} (0.18), the study essentially followed the American Thoracic Society (ATS) and European Community for Coal and Steel (ECCS) recommendations for standardizing the methodology of measuring D_{LCO}. Prediction equations for ages 20 through 70 were calculated separately for both sexes. Simple linear equations using age, height, and body weight as independent variables predicted the D_{LCO} indices (D_{LCO}, V_A, and D_L/V_A) as well as more complex equations. In addition, a complete analysis of the residuals (predicted measured values) showed that the assumptions of the multiple regression analysis (independence, homoscedasticity, and Gaussian distribution of residuals) were fulfilled using simple linear equations. Correction for the instrumental and anatomic dead spaces decreased the D_{LCO} as average of 4.7% The standard error of estimates was lower than those reported from other series in the literature. The predicted values from this study were lower than those reported by some investigators and were in reasonable agreement with other studies. A portion but not all of the differences could be explained on the basis of recognized differences in testing methodology. The results of this study may be of value to clinical laboratories seeking predictive equations for D_{LCO} most appropriate for their testing methodology and patient population, and may assist in the resolution of some controversies regarding differences among predictive equations for D_{LCO}.

Attenuation of the Metabisulfite-Induced Bronchoconstrictive Response by Pretreatment with Cromolyn—MD McClellan, JS Wanger, RM Cherniack. Chest 1990;97:826.

In a retrospective analysis of 1544 patients who underwent provocative challenge with metabisulfite at the National Jewish Center between 1983 and 1987, an abnormal airway response to metabisulfite was found in 52 patients, an incidence of 3.4%. There was no relationship between this abnormal airway responsiveness to metabisulfite and the degree of airway obstruction present or the degree of airway reactivity as assessed by the response to inhaled bronchodilator or exercise testing. In a pilot study, we found that the administration of cromolyn sodium prior to metabisulfite challenge markedly attenuated the abnormal bronchoconstrictive response in 9 of 10 patients. We conclude that a metabisulfite-induced bronchoconstrictive response cannot be predicted on the basis of the degree of airway obstruction or airway reactivity and that pretreatment with cromolyn sodium may attenuate the abnormal response.

Factors Related to the Nocturnal Worsening of Asthma—RJ Martin, LC Cicutto, RD Ballard. Am Rev Respir Dis 1990;141:33.

The nocturnal worsening of asthma is a very common problem, yet little is known about the relationships between the nocturnal worsening and daytime lung function, methacholine bronchial responsiveness, the degree of the circadian variability in bronchial responsivity, and the nocturnal sleep pattern. This study demonstrates in 20 asthmatic patients that the overnight fall in the peak expiratory flowrates (PEFR) is related to the severity of daytime airflow limitation ($r = 0.73$, $p < 0.001$) and daytime bronchial responsiveness ($r = 0.48$, $p < 0.05$). In individuals with larger overnight decrements in PEFR, bronchial responsivity at 0400 h is so great that normal saline inhalation alone can produce a < 20% fall in the FEV_1. Sleep quality and sleep staging are not correlated to the change in the PEFR. Thus, the overnight decrement in asthmatic lung function is related to the daytime severity of asthma as determined by daytime measurements

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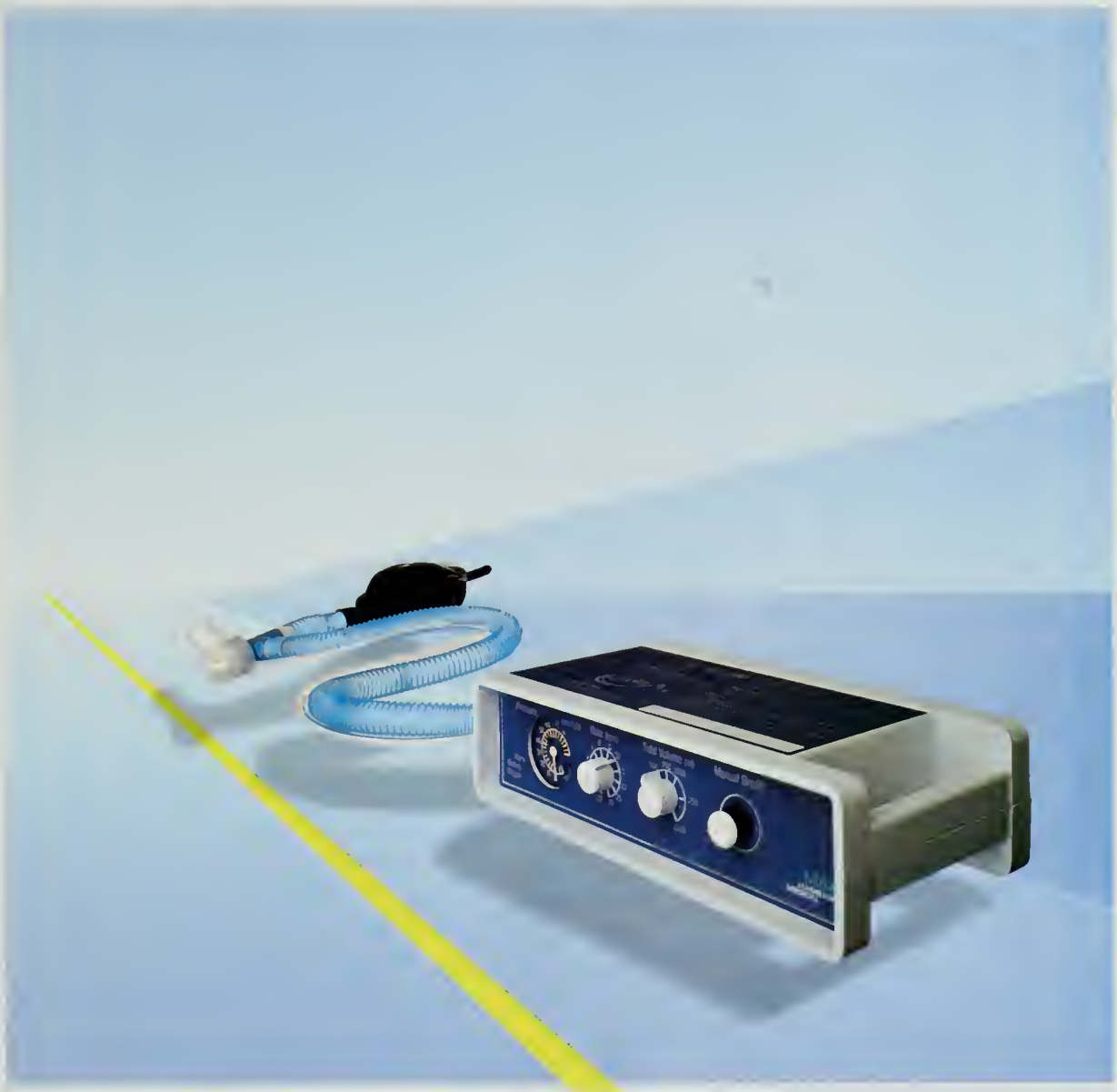

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of airflow limitation and bronchial responsiveness as well as the circadian variation in bronchial responsivity.

Apnea, Transient Episodes of Bradycardia, and Periodic Breathing in Preterm Infants—JE Hodgman, F Gonzalez, T Hoppenbrouwers, LA Cabal. AJDC 1990;144:54.

The occurrence of central apnea of 15 s or longer, transient episodes of bradycardia (TEB), and periodic breathing were studied in 66 healthy premature infants when at least 1 week old and between 32 and 36 weeks postconceptual age. Eight-hour cardiorespiratory recordings were visually scanned for the presence of these patterns. Central apnea of 15 s or longer was seen in almost half of the infants. The TEB were numerous, and the majority were not associated with central apnea; however, all but five of the apneic episodes that lasted 15 s or longer were accompanied by TEB. Infants spent as much as 40% of their time in periodic breathing. The frequency with which these patterns are seen in healthy premature infants strongly suggests that they are normal findings. Our results do not support the opinion that brief periods of apnea are abnormal when accompanied by TEB.

Oxygen Desaturation during Sleep as a Function of the Underlying Respiratory Disease—B Midgren. Am Rev Respir Dis 1990;141:43.

Patients with hypoxemia due to respiratory diseases may desaturate further during sleep. A quantitative analysis of this sleep-induced desaturation, simultaneously comparing patients with different respiratory diseases, could identify patient groups especially prone to develop nocturnal hypoxemia. I have therefore compared the changes in oxygenation during sleep in three groups of patients with the same baseline oxygen saturation

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BRIEF SUMMARY

***Potency expressed as albuterol.**

The following is a brief summary only. Before prescribing, see complete prescribing information in Ventolin® Solution for Inhalation product labeling.

CONTRAINDICATIONS: Ventolin® Solution for Inhalation is contraindicated in patients with a history of hypersensitivity to any of the components.

WARNINGS: As with other inhaled beta-adrenergic agonists, Ventolin® Solution for Inhalation can produce paradoxical bronchospasm that can be life-threatening. If it occurs, the preparation should be discontinued immediately and alternative therapy instituted.

Fatalities have been reported rarely in association with the use of inhaled sympathomimetic drugs and with the home use of nebulizers. It is therefore essential that the physician instruct the patient in the need for further evaluation if his/her asthma becomes worse. In individual patients, any $beta_2$-adrenergic agonist, including albuterol solution for inhalation, may have a clinically significant cardiac effect.

Immediate hypersensitivity reactions may occur after administration of albuterol, as demonstrated by rare cases of urticaria, angioedema, rash, bronchospasm, and oropharyngeal edema.

PRECAUTIONS: General: Albuterol, as with all sympathomimetic amines, should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias, and hypertension, in patients with convulsive disorders, hyperthyroidism, or diabetes mellitus, and in patients who are unusually responsive to sympathomimetic amines.

Large doses of intravenous albuterol have been reported to aggravate pre-existing diabetes mellitus and ketoacidosis. Additionally, beta agonists, including albuterol, given intravenously may cause a decrease in serum potassium, possibly through intracellular shunting. The decrease is usually transient, not requiring supplementation. The relevance of these observations to the use of Ventolin® Solution for Inhalation is unknown.

Information for Patients. The action of Ventolin Solution for Inhalation may last up to six hours, and therefore it should not be used more frequently than recommended. Do not increase the dose or frequency of medication without medical consultation. If symptoms get worse, medical consultation should be sought promptly. While taking Ventolin Solution for Inhalation, other antiasthma medicines should not be used unless prescribed.

See illustrated Patient's Instructions for Use section of the package insert.

Drug Interactions. Other sympathomimetic aerosol bronchodilators or epinephrine should not be used concomitantly with albuterol.

Albuterol should be administered with extreme caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants because the action of albuterol on the vascular system may be potentiated.

Beta-receptor blocking agents and albuterol inhibit the effect of each other.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Albuterol sulfate, like other agents in its class, caused a significant dose-related increase in the incidence of benign leiomyomas of the mesovarium in a two-year study in the rat, at oral doses corresponding to 10, 50, and 250 times the maximum human nebulizer dose. In another study, this effect was blocked by the coadministration of propranolol. The relevance of these findings to humans is not known. An 18-month study in mice and a lifetime study in hamsters revealed no evidence of tumorigenicity. Studies with albuterol revealed no evidence of mutagenesis. Reproduction studies in rats revealed no evidence of impaired fertility.

Pregnancy: *Teratogenic Effects*; *Pregnancy Category C*: Albuterol has been shown to be teratogenic in mice when given subcutaneously in doses corresponding to the human nebulization dose. There are no adequate and well-controlled studies in pregnant women. Albuterol should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. A reproduction study in CD-1 mice with albuterol (0.025, 0.25, and 2.5 mg/kg subcutaneously, corresponding to 0.1, 1, and 12.5 times the maximum human nebulization dose, respectively) showed cleft palate formation in 5 of 111 (4.5%) of fetuses at 0.25 mg/kg and in 10 of 108 (9.3%) of fetuses at 2.5 mg/kg. None was observed at 0.025 mg/kg. Cleft palate also occurred in 22 of 72 (30.5%) fetuses treated with 2.5 mg/kg isoproterenol (positive control). A reproduction study in Stride Dutch rabbits revealed cranioschisis in 7 of 19 (37%) fetuses at 50 mg/kg, corresponding to 250 times the maximum human nebulization dose.

Labor and Delivery: Oral albuterol has been shown to delay preterm labor in some reports. There are presently no well-controlled studies that demonstrate that it will stop preterm labor or prevent labor at term. Therefore, cautious use of Ventolin® (albuterol sulfate, USP) Solution for Inhalation is required in pregnant patients when given for relief of bronchospasm so as to avoid interference with uterine contractility.

Nursing Mothers. It is not known whether this drug is excreted in human milk. Because of the potential for tumorigenicity shown for albuterol in some animal studies, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use: Safety and effectiveness in children below 12 years of age have not been established.

ADVERSE REACTIONS: The results of clinical trials with Ventolin® Solution for Inhalation in 135 patients showed the following side effects that were considered probably or possibly drug-related.

Central Nervous System. Tremors (20%), dizziness (7%), nervousness (4%), headache (3%), insomnia (1%).

Gastrointestinal. Nausea (4%), dyspepsia (1%).

Ear, Nose, and Throat: Pharyngitis (<1%), nasal congestion (1%).

Cardiovascular: Tachycardia (1%), hypertension (1%).

Respiratory. Bronchospasm (8%), cough (4%), bronchitis (4%), wheezing (1%).

No clinically relevant laboratory abnormalities related to Ventolin Solution for Inhalation administration were determined in these studies.

The following moderate to severe reactions, as judged by the investigators, were reported in three-month clinical trials. Mild reactions are not included.

Central Nervous System. Tremor (10.7%), headache and insomnia (3.1% each).

Cardiovascular: Hypertension (3.1%). Although arrhythmias and palpitation were not reported for Ventolin Solution for Inhalation, this should not be interpreted as indicating that these adverse events cannot occur after the administration of inhaled albuterol.

Respiratory: Bronchospasm, generally exacerbations in the underlying pulmonary disease, (15.4%), cough (3.1%), bronchitis, wheezing, sputum increase, dyspnea (1.5%) each.

Gastrointestinal: Nausea (3.1%), dyspepsia (1.5%).

Systemic: Malaise (1.5%).

Rare cases of urticaria, angioedema, rash, bronchospasm, and oropharyngeal edema have been reported after the use of inhaled albuterol.

OVERDOSAGE: Information concerning possible overdosage and its treatment appears in the full prescribing information.

DOSAGE AND ADMINISTRATION: The usual dosage for adults and children 12 years and older is 2.5 mg of albuterol administered three to four times daily by nebulization. More frequent administration or higher doses are not recommended. To administer 2.5 mg of albuterol, dilute 0.5 ml of the 0.5% solution for inhalation with 2.5 ml of sterile normal saline solution. The flow rate is regulated to suit the particular nebulizer so that Ventolin® Solution for Inhalation will be delivered over approximately five to 15 minutes.

The use of Ventolin Solution for Inhalation can be continued as medically indicated to control recurring bouts of bronchospasm. During this time most patients gain optimal benefit from regular use of the solution for inhalation.

If a previously effective dosage regimen fails to provide the usual relief, medical advice should be sought immediately as this is often a sign of seriously worsening asthma that would require reassessment of therapy.

HOW SUPPLIED: Ventolin® Solution for Inhalation, 0.5% is supplied in bottles of 20 ml (NDC 0173-0385-58) with accompanying calibrated dropper in boxes of one.

Store between 2° and 30°C (36° and 86°F).

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but with entirely different types of respiratory dysfunction, namely interstitial lung disease (n = 14), chronic obstructive pulmonary disease (n = 29), and scoliosis (n = 10). The patients with scoliosis had considerably larger sleep-induced desaturation than the patients with lung diseases. Their oxygen saturation was also more unstable than that of the others during REM sleep—possibly a consequence of their lower position on the oxyhemoglobin dissociation curve during this sleep stage. The results suggest that sleep studies may be more informative in patients with scoliosis, for example, where a need for nocturnal-assisted ventilation can be revealed, than in patients with lung diseases.

A Pilot Study of Aerosolized Amiloride for the Treatment of Lung Disease in Cystic Fibrosis—MR Knowles, NL Church, WE Waltner, et al. N Engl J Med 1990;322:1189.

Excessive active absorption of sodium is a unique abnormality of the airway epithelium in patients with cystic fibrosis. This defect is associated with thickened mucus and poor clearance of airway secretions and may contribute to the pulmonary disease in these patients. To study whether the inhibition of excessive absorption of sodium might affect the course of lung disease in cystic fibrosis, we performed a double-blind, crossover trial comparing aerosolized amiloride (5 mmol/L; 3.5 mL four times daily), a sodium-channel blocker, with vehicle alone. Fourteen of the 18 adult patients initially enrolled in the study completed the one-year trial (25 weeks for each treatment). The mean (± SEM) loss of forced vital capacity (FVC) was reduced from 3.39 ± 1.13 mL per day during treatment with vehicle alone to 1.44 ± 0.67 mL per day during treatment with amiloride ($p < 0.04$). A measured index of sputum viscosity and elasticity was abnormal during treatment with vehicle alone and improved during treatment with amiloride. Calculated indexes of mucociliary and cough clearance also improved during amiloride treatment. No systemic, respiratory or subjective toxic effects of amiloride were noted. We conclude from this preliminary study that aerosolized amiloride can be safely administered to adults with cystic fibrosis. The slowing of the loss of FVC and the improvement in sputum viscosity and elasticity suggest a beneficial clinical effect. Aerosolized amiloride deserves further evaluation in the treatment of lung disease in patients with cystic fibrosis.

The Bronchodilator Response from Increasing Doses of Terbutaline Inhaled from a Multi-Dose Powder Inhaler (Turbuhaler)—G Persson, JE Wirén. Eur Respir J 1990;3:24.

Twelve adult asthmatics inhaled single doses of 0.5, 2.0, or 4.0 mg of terbutaline, respectively, via Turbuhaler, on three separate days. Forced expiratory volume in one second (FEV_1) forced vital capacity (FVC), and pulse rate were measured 15, 30, 45, and 60 min after inhalation from the inhaler and then hourly up to 8 h. The results showed that a more rapid onset of action, a greater maximal response, and a longer duration of action can be achieved by exceeding the standard dose of terbutaline of 0.5 mg. The incidence of adverse effects was very low even at the highest dose, 4 mg. This study emphasizes the need for individual dosing of inhaled bronchodilators.

Agreement between Noninvasive Oximetric Values for Oxygen Saturation—C Cahan, MJ Decker, PL Hoekje, KP Strohl. Chest 1990;97:814.

We made an assessment of five pulse oximeters in regard to their ability to relpace the Hewlett-Packard (HP) ear oximeter as a noninvasive measurement of S_{aO_2}. Trials were performed during isocapnic progressive hypoxia (S_{aO_2} range, 99 to 70%) in 22 white and 6 black subjects. Comparisons between values of S_{aO_2} by oximetry were analyzed by comparing the difference of values by the two methods against their mean. The difference between pulse oximeters and the HP was 2.6 ± 10.3% in all subjects. Results in whites were 1.9 ± 10.2% and in blacks were 5.1 ± 9.2%. The distribution of differences between pulse oximeters and the HP were larger below 80% than above 85%. We conclude that pulse oximeters give higher values than the HP, a tendency that is more pronounced in black than in white subjects. While the limits of agreement are better at saturations above 85%, the 95% confidence limits of agreement between pulse oximeters and the HP are rather large (± 10%) and unacceptable for assuming that pulse oximeters will provide the same values as found in clinical studies using the HP.

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Adult Respiratory Distress Syndrome in Severely Neutropenic Children—Y Sivan, C Mor, S Al-Jundi, CJL Newth. Pediatr Pulmonol 1990;8:104.

It has been suggested that polymorphonuclear cells (PMNs) are required for the development of the adult respiratory distress syndrome (ARDS). We investigated the occurrence of ARDS with acute respiratory failure in 30 children with severe neutropenia ($<$ 500 PMNs/mm^3) who met the clinical diagnostic criteria for ARDS and in whom postmortem histopathology findings were available within 7 days of the onset of ARDS. In 26 patients, the histopathology was consistent with ARDS. In 12/26 children no white blood cells (WBC) were found in the lung tissue, 10/26 had moderate infiltration of mononuclear cells, 2/26 had massive tumor cell infiltration, and in 2/26 PMNs were found. Thus, in at least 22/26 patients ARDS developed without neutrophilic infiltration of the lungs. The maximum active lung infection rate was found to be 69% (18/26) by endotracheal and postmortem lung cultures and histology. Thus 5/26 children had ARDS without any WBC in the lung tissue. We conclude, as have other studies in adults, that the absence of PMNs does not protect children from the development of ARDS and that the mechanism that involves PMNs is probably only one of several pathways to diffuse alveolar damage, some of which is neutrophil-independent.

Effects of Nasal CPAP on Supraglottic and Total Pulmonary Resistance in Preterm Infants—MJ Miller, JA DiFiore, KP Strohl, RJ Martin. J Appl Physiol 1990;68:141.

The effects of continuous positive airway pressure (CPAP) on supraglottic and total pulmonary resistance were determined in 10 healthy premature infants (postconceptional age 34 $\pm$ 2 wk, weight at study 1628 $\pm$ 250 g). Nasal airflow was measured with a mask pneumotachograph, and pressures in the esophagus and oropharynx were measured with a 5-Fr Millar or fluid-filled catheter. Nasal CPAP between 0 and 5 cm H_2O correlated well with oropharyngeal pressure ($r = 0.94$) Total supraglottic resistance, total pulmonary resistance, and supraglottic resistance in inspiration and expiration were measured on increasing CPAP. Total supraglottic resistance decreased from 46 $\pm$ 29 to 17 $\pm$ 16 cm $H_2O \cdot L^{-1} \cdot s$ ($p < 0.005$) between 0 and 5 cm H_2O CPAP, and a delay in return of resistance to control values was seen as CPAP was reciprocally decreased to 0. CPAP produced a decrease in supraglotttic resistance in both inspiration and expiration from 41 $\pm$ 26 to 14 $\pm$ 9 and from 33 $\pm$ 17 to 10 $\pm$ 6 cm $H_2O \cdot L^{-1} \cdot s$, respectively ($p < 0.01$). Total pulmonary resistance also decreased from 161 $\pm$ 40 to 95 $\pm$ 24 cm $H_2O \cdot L^{-1} \cdot s$, ($p < 0.01$) between 0 and 5 cm H_2O CPAP. The decrease in total supraglottic resistance in these infants accounted for 60% of the change in total pulmonary resistance, which occurred on CPAP of 5 cm H_2O. We speculate that CPAP may decrease supraglottic resistance directly through mechanical splinting of the airway. This effect of CPAP may be the primary mechanism by which this form of therapy reduces apnea with an obstructive component in premature infants.

Continuous Positive Airway Pressure Reduces Work of Breathing and Dyspnea during Weaning from Mechanical Ventilation in Severe Chronic Obstructive Pulmonary Disease—BJ Petrof, M Legaré, P Goldberg, J Milic-Emili, SB Gottfried. Am Rev Respir Dis 1990;141:281.

Dynamic hyperinflation and the development of intrinsic positive end-expiratory pressure ($PEEP_i$) are commonly observed in patients with severe chronic obstructive pulmonary disease (COPD) and acute respiratory failure. The presence of $PEEP_i$ acts as an inspiratory threshold load, and contributes significantly to the observed increase in work and oxygen cost of breathing. The present study examined the effects of continuous positive airway pressure (CPAP) (at 5, 10, and 15 cm H_2O) and its ability to reduce the mechanical load imposed by $PEEP_i$ on breathing pattern, work of breathing, and dyspnea in seven patients with severe COPD during weaning from mechanical ventilation. Tidal volume remained stable at all levels of applied pressure. Breathing frequency was also stable except for a small (12%) decrease during CPAP of 15 cm H_2O. Inspiratory pulmonary resistance and elastance were unaltered by the application of CPAP. There were progressive reductions in the inspiratory work of breathing as the level of CPAP increased. At the highest level of CPAP, the amount of inspiratory work performed per minute and per liter of ventilation decreased by 49.8 and 41.8%, respectively. Similar progressive reductions were also obtained in the pressure-time product for the inspiratory muscles and the diaphragm, which amounted to decreases of 42.9 and 42.2%, respectively, at the highest level of CPAP. End-expiratory lung volume remained stable at the lowest level of CPAP, with only modest increases occurring at the higher levels. In addition, all patients reported a reduction in dyspnea during the administration of CPAP. We conclude that CPAP, by reducing the inspiratory mechanical load, decreases the inspiratory work of breathing and sense of breathlessness in patients with severe COPD and acute respiratory failure. This may be of potential therapeutic benefit during weaning from mechanical ventilation as well as during acute exacerbations of severe COPD in the nonintubated patient.

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Depressed Bronchoalveolar Urokinase Activity in Patients with Adult Respiratory Distress Syndrome—P Bertozzi, B Astedt, L Zenzius, et al. N Engl J Med 1990;322:890.

Abundant deposition of bronchoalveolar fibrin and fibronectin occurs during the exudative phase of the adult respiratory distress syndrome (ARDS), promoting hyaline-membrane formation and subsequent alveolar fibrosis. To explore the mechanisms that account for the persistence of bronchoalveolar fibrin and fibronectin, we compared the activity of urokinase, which is necessary for plasminogen activation and fibrin degradation, in cell-free bronchoalveolar-lavage fluid from 8 patients with ARDS, 9 patients with acute pulmonary diseases other than ARDS, and 10 normal subjects. The mean level of urokinase activity in the lavage fluid from the patients with ARDS was 0.003 IU per mL of fluid (range, 0 to 0.008), which was significantly lower ($p = 0.001$) than the level in the fluid from either the patients with pulmonary diseases other than ARDS (0.118 IU per mL [range, 0.032 to 0.295]) or the normal subjects (0.129 IU per mL [range, 0.045 to 0.198]). The lavage fluid from all the patients with ARDS also had antiplasmin activity, which would promote the persistence of fibrin. A true decrease in urokinase activity was confirmed by the failure of the lavage fluid from the patients with ARDS to convert [^{125}I] plasminogen to plasmin. Despite the low urokinase activity, immunochemical assays revealed normal levels of urokinase antigen in the fluid from the patients with ARDS, suggesting the presence of urokinase inhibitors. Inhibitors were demonstrated directly by a fibrin gel-underlay assay that detects complexes of urokinase with inhibitors. Plasminogen-activator inhibitor type 1 was the principal inhibitor identified. We conclude that increased antifibrinolytic activity due to both urokinase inhibitors and antiplasmins in the bronchoalveolar compartment of patients with ARDS contributes to the formation and persistence of hyaline membranes, a key component of alveolar histopathology in ARDS.

Sleep Disturbanees in Patients with Asthma—C Janson, T Gislason, G Boman, J Hetta, B-E Roos—Respir Med 1990;84:37.

The prevalence of sleep complaints and sleep disturbances was studied prospectively in 98 consecutive adults asthmatic patients (mean age 45 Years, 46% men) attending an outpatient clinic, by means of questionnaires and sleep diaries. The results were compared with those from an age- and sex-matched group of 226 healthy individuals. The most common sleep disturbances among the asthmatic patients were early morning awakening (51%), difficulty in maintaining sleep (DMS;44%) and daytime sleepiness (44%). With decreasing asthma control (ie, increased number of acute asthmatic attacks) there was an increase of DMS, nocturnal wakefulnes, nocturnal breathing problems and bronchodilator inhalations at night. A decrease in estimated sleep time ($p < 0.05$) and increase in nocturnal wakefulness ($p < 0.01$) was seen with decreasing daytime FEV_1—measured as percentage of the predicted value ($\%FEV_1$) There was also significant correlation between increasing age and decreasing $\%FEV_1$ ($p < 0.01$). Among the 26 patients who were only taking one oral bronchodilator, no definite difference regarding sleep quality was found between those treated with theophylline and those taking an oral β_2 agonist. The prevalence rates of DIS, DMS, and daytime sleepiness were about twice as high among the asthmatic patients than in the healthy population. It is concluded that impaired quality of sleep, with disturbed sleep during the night, early morning awakenings, and daytime sleepiness, is common among patients with bronchial asthma.

Breath-Stacking Increases the Depth and Duration of Chest Expansion by Incentive Spirometry—WL Baker, VJ Lamb, JJ Marini. Am Rev Respir Dis 1990;141:343.

Although the objective of incentive spirometry is to achieve and hold high lung volumes, many patients with pain or weakness are unable to sustain the effort needed to perform effective exercises. We questioned whether using a one-way valve to prevent exhalation would allow rest between inspiratory efforts and cause volume to cumulate during successive tidal efforts, improving both the depth and duration of the inspiratory maneuver. We studied 26 cooperative but naive patients recovering from surgery, trauma, or critical illnes whose pain or weakness impaired ability to achieve and sustain deep inspiration. All subjects breathed via mouthpiece from a spirometer prefilled with 100% oxygen. Three different maneuvers were performed in random order by all subjects: (1) standard inspiratory capacity without valve or inspiratory hold, (2) inspiratory capacity (IC) with breathholding aided by a one-way valve, and (3) uncoached breath-stacking, during which successive tidal breaths were cumulated by one-way valving. A fourth maneuver was added in the last 13 subjects studied: an initial coached IC effort with subsequent valved stacking of tidal efforts. When compared with IC, "breath-stacking" (valved) maneuvers increased inspired volume by an average of 15 to 20% ($p < 0.05$). More importantly, there was a severalfold increase in the time over which high lung volume was sustained ($p < 0.001$). Our results indicate that one-way valving helps to achieve and sustain deep inspiration, even in uncoached patients.

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Cardiac Arrhythmias during Exercise in Severe Chronic Obstructive Pulmonary Disease—TH Cheong, S Magder, S Shapiro, JG Martin, RD Levy. Chest 1990;97:793.

We studied the effect of maximal exercise on the prevalence of arrhythmias in 122 subjects with severe COPD. At rest, ten subjects had supraventricular arrhythmias while 13 had unifocal ventricular premature beats $\geqslant 6$/min or ventricular bigeminy. At peak exercise, six subjects had supraventricular arrhythmias while 24 had ventricular arrhythmias. Univariate and multivariate analysis with logistic regression did not show relationships between exercise-related cardiac arrhythmias and the severity of pulmonary disease, oxyhemoglobin desaturation or ECG evidence of chronic lung disease. Exercise-related arrhythmias were significantly associated with the presence of arrhythmias at rest and 87% of subjects who had no arrhythmias at rest did not have any during exercise. In patients with COPD, the development of potentially serious arrhythmias during exercise is uncommon without clinically apparent CAD or arrhythmias at rest. However, routine cardiac monitoring during exercise testing should not be abandoned in this population since ventricular arrhythmias can occur despite their absence at rest.

Observations on Intracranial Dynamics during Respiratory Physiotherapy in Unconcious Neurosurgical Patients—U Ersson, H Carlson, A Mellström, U Pontén, U Hedstrand, S Jakobsson. Acta Anaesthesiol Scand 1990;34:99.

The effects of airway care procedures on intracranial dynamics were studied in 12 patients with intracranial lesions. The patients had controlled ventilation and were treated according to a standardized protocol with endotracheal suction and bag squeezing. Intracranial, arterial blood, and airway pressures were recorded and cerebral perfusion pressure was calculated. Both methods used for airway care elicited marked changes in intracranial, arterial blood and cerebral perfusion pressures during the treatment session. However, within 1 min after termination of the procedure the different pressure levels returned to pretreatment values except for the intracranial and arterial blood pressure in endotracheal suction. Cerebral perfusion pressure, calculated at different time intervals: 1, 5, and 15 min after the treatment, showed only minor deviations from values before treatment. The mean values varied from 70 to 90 mm Hg (9.3 to 12.0 kPa) and the lower limit of the 99% confidence interval of the means was never below 55 mm Hg (7.3 kPa). At times complementary administration of sedatives during endotracheal suction was found to induce a drop in arterial blood pressure and cerebral perfusion pressure. In conclusion, patients with severe brain injuries treated on mechanical ventilation are exposed to equal risks when using bag squeezing for airway care as when using traditional endotracheal suction. Regardless of the method used, patients should be adequately sedated before starting procedure in order to reduce the risk of adverse effects.

Thoracoabdominal Asynchrony in Infants with Airflow Obstruction—JL Allen, MR Wolfson, K McDowell, TH Shaffer, with technical assistance of Raymond Motley. Am Rev Respir Dis 1990;141:337.

Thoracoabdominal asynchrony (TAA) has long been thought clinically useful in the assessment of airflow obstruction (AO) in infants. To test the hypothesis that the measurement of TAA is useful in the assessment of lung mechanics in infants with AO, we have used respiratory inductive plethysmography (RIP) to quantify TAA. We compared changes in TAA to changes in lung mechanics before and after aerosolized bronchodilator (BD) administration in 13 infants. Abdominal wall (AB) and rib-cage (RC) motion were displayed on an X-Y recorder in a Lissajous figure. Asynchrony RC and AB motion was quantified by comparing the width m of the Lissajous figure (difference between AB inspiratory and expiratory positions) at mid-RC excursion with the total AB excursion at its extremes (s). Phase angle ϕ was computed as $\sin \phi = m/s$ (or $\phi = 180° - \mu$, where $\sin \mu = m/s$ for phase angles $> 90°$) and was taken as a measure of TAA. Lung resistance (RL) and elastance (EL) were calculated from esophageal pressure (Pes), mouth pressure, tidal volume, and tidal flow. All infants displayed TAA at baseline. After BD administration, TAA decreased in those infants in whom RL decreased. The percentage decrease in the phase angle from baseline after BD administration was significantly correlated with the decrease in peak-to-peak Pes (Δ) and the percentage decrease in RL and EL. We conclude that AO in infants leads to TAA through altered pleural pressure swings acting on the compliant chest wall. Changes in lung mechanics induced by bronchodilators are reflected in changes in TAA. The quantification of TAA may be a useful indicator of infant lung function.

Cricopharyngeal Dysfunction in Chronic Obstructive Pulmonary Disease—M Stein, AJ Williams, F Grossman, AS Weinberg, L Zuckerbraun. Chest 1990;97:347.

Dysphagia due to cricopharyngeal dysfunction is well known; however, there have been no previous data indicating an association between cricopharyngeal dysfunction and COPD. After observing marked cricopharyngeal dysfunction with aspiration

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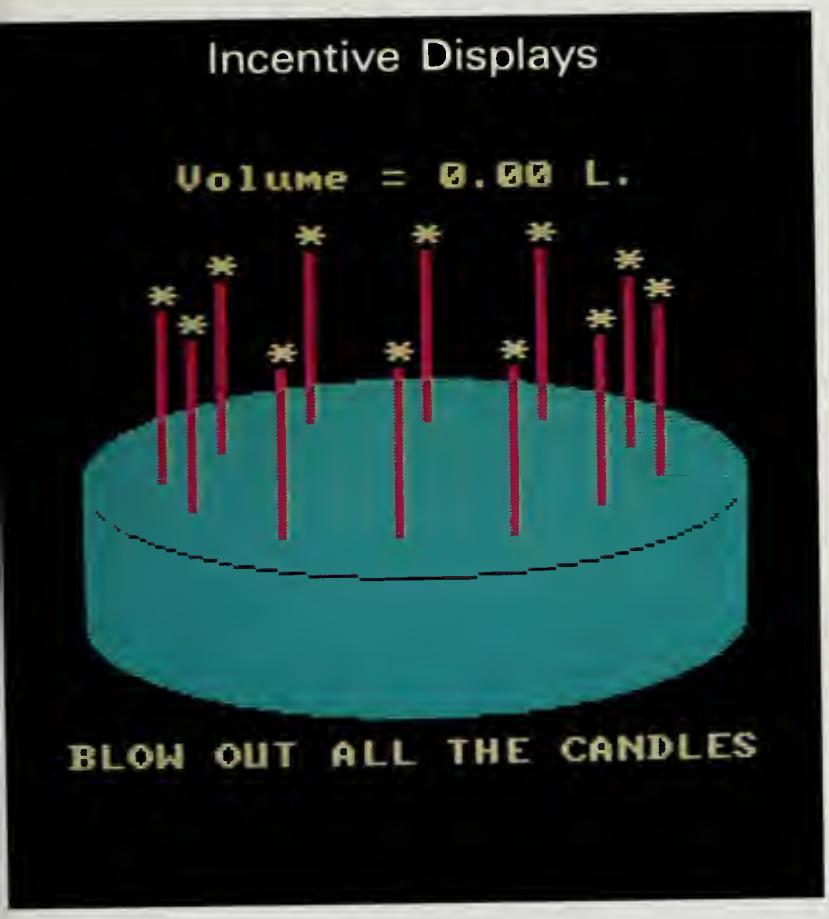

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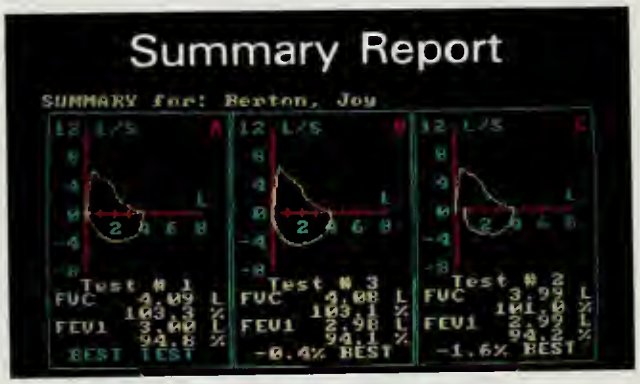

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in three patients who had frequent and severe exacerbations of COPD, we performed pharyngoesophageal examinations with videotaping in another 22 nonrandomized patients. Cineradiography or videofluoroscopic recording with capabilities of slow-motion and freeze-frame playback is mandatory, since the transit time of the bolus through the pharynx is rapid. Severe cricopharyngeal dysfunction was observed in 17 elderly patients with COPD. Deglutition disorders were elicited by careful questioning in 15 of these. In 8 subjects, cricopharyngeal myotomy resulted in improvement of swallowing and complete or partial relief of acute exacerbations of respiratory distress. In one subject, myotomy relieved only the swallowing problem. The mechanism of cricopharyngeal dysfunction in elderly patients with COPD is unknown at this time, but may be related to gastroesophageal reflux, therapeutic agents, and/or alterations in pharyngoesophageal anatomic structures. We conclude that investigations for swallowing disorders should be considered in patients with COPD who have frequent acute exacerbations of respiratory distress.

Cross-Sectional Echocardiographic Characterization of Atelectatic Lung Segments: Differentiation from Extracardiac Tumors—DL Mann, K Thompson, J Kaiser. Chest 1990;97:404.

This report describes a unique series of patients who, during routine cross-sectional echocardiographic examination, were each noted to have a large echo-dense extracardiac mass adherent to the lateral aspect of the left ventricle. While this echo-dense mass was considered intitially to represent an extracardiac tumor, this mass was shown subsequently to be an atelectatic segment of the left lower lobe of the lung. The salient echocardiographic findings that were considered to be helpful in terms of differentiating these adherent pulmonary atelectatic lung segments were that the lung segments always occurred in the presence of a moderate-to-large left pleural effusion; in real-time examination, the atelectatic lung masses generally appeared solid, as opposed to cystic, with a characteristic brightly reflective ground-glass appearance; there was never any evidence of extrinsic compression of the heart by the lung mass.

Use of Extracorporeal Membrane Oxygenation in the Treatment of Respiratory Syncytial Virus Bronchiolitis: The National Experience, 1983 to 1988—RH Steinhorn, TP Green. J Pediatr 1990;116:338.

In an effort to obtain data to provide the basis for the design of controlled clinical trials, we contacted all U.S. participants in the National ECMO Registry to assemble the national experience on the use of ECMO in respiratory syncytial virus bronchiolitis during the past 5 years. Twelve infants were treated at nine centers between 1983 and 1988. Eight had been born prematurely, and five had bronchopulmonary dysplasia. The mean age at onset of infection with respiratory syncytial virus was 108 $\pm$ 102 days. The mean length of ventilator management before ECMO was 7.8 $\pm$ 7.1 days. All infants had persistent hypoxemia with a mean arterial oxygen pressure of 39.2 $\pm$ 11.7 torr (5.3 $\pm$ 1.6 kPa) despite high ventilator pressures (mean airway pressure 19.7 $\pm$ 6.4 cm H_2O) and 100% inspired oxygen; six had air leak syndrome. Seven infants survived (58%). The mean duration of ECMO for survivors was 233 $\pm$ 139 hours. Pre-existing chronic lung disease did not predict a poor outcome: Four of the five infants with bronchopulmonary dysplasia survived. Six of the survivors have subsequently achieved expected developmental milestones and one has slight motor delay. We conclude that, for infants with severe respiratory syncytial virus bronchiolitis whose condition deteriorates despite maximal ventilator management, ECMO may provide lifesaving support. The duration of successful treatment with this therapy may be longer than that for conventional neonatal indications, but excellent neurologic outcome may be expected in survivors.

Relationship between Infant Lung Mechanics and Childhood Lung Function in Children of Very Low Birthweight—KN Chan, YC Wong, and M Silverman. Pediatr Pulmonol 1990;8:74.

Twenty-seven children of very low birthweight ($\leqslant$ 1,500 g) whose lung function had been measured on several occasions during the first year were studied at the age of about 9 years. Fifteen of the children had received neonatal intermittent positive pressure ventilation, mostly for respiratory distress syndrome. Ten of the ventilated children were still oxygen dependent at 30 days of age. Compared to the remainder of the group, mechanically ventilated children had reduced lung compliance in early infancy and increased thoracic gas volume in the middle of their first year. These changes correlated with the level of neonatal respiratory therapy as indicated by the oxygen score. Lung compliance in early infancy, but not thoracic gas volume, correlated with forced expiratory volume at 1 second recorded at 9 years. On the other hand, reduced airway conductance showed no significant correlation with the neonatal oxygen score, but there was a strong correlation between airway conductance late in infancy and lung function at 9 years. This relationship was independent of neonatal mechanical ventilation. We conclude that perinatal factors, which may be associated with

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disturbed lung mechanics early in infancy, are only weak and indirect predictors of childhood lung function. Airway conductance late in infancy, determined by constitutional factors, prematurity itself, or other undetermined factors, is a good predictor of airway function at 9 years.

'Common Courtesy' and the Elimination of Passive Smoking: Results of the 1987 National Health Interview Survey—RM Davis, GM Boyd, CA Schoenborn. JAMA 1990;263: 2208.

The tobacco industry recommends "common courtesy" as the solution to potential conflicts over smoking in public places and as an alternative to policies that restrict or ban smoking. Specifically, the industry suggests that nonsmokers "mention annoyances in a pleasant and friendly manner" and that smokers ask others, "Do you mind if I smoke?" We analyzed data for 22,000 adults who responded to the 1987 National Health Interview Survey of Cancer Epidemiology and Control to determine if common courtesy is being used in passive-smoking situations. Almost half (47%) of smokers said they light up inside public places without asking if others mind. When someone lights up a cigarette inside a public place, only 4% of nonsmokers ask the person not to smoke despite the fact that most nonsmokers consider secondhand smoke harmful and annoying. We compared these data with similar data collected by the Roper Organization in the 1970s and found that smokers today are less likely to smoke inside public places. However, nonsmokers' actions in response to secondhand smoke have changed very little. These findings show that the common courtesy approach endorsed by the tobacco industry is unlikely, by itself, to eliminate exposure to environmental tobacco smoke. Though no one would oppose the use of common courtesy, we conclude that legislative or administrative mechanisms are the only effective strategies to eliminate passive smoking.

Pulmonary Wegener's Granulomatosis: A Clinical and Imaging Study of 77 Cases—J-F Cordier, D Valeyre, L Guillevin, R Loire, J-M Brechot. Chest 1990;97:906.

We studied 77 patients with biopsy-proven Wegener's Granulomatosis (WG) and pulmonary manifestations, to characterize the nature and frequency of the clinical, imaging, and endoscopic features of this condition. Pulmonary symptoms were cough, mild dyspnea, hemoptysis, and chest pain. Five patients had no pulmonary symptoms. Imaging features consisted of nodules, infiltrates, and pleural opacities, which were not seen on an x-ray film. Fiberoptic bronchoscopy was performed in 74 patients, and it was macroscopically abnormal in 55% (showing bronchial inflammation or stenosis or both or isolated hemorrhage). Six patients presented with alveolar hemorrhagic syndrome. Four patients had a pleural exudate rich in polymorphonuclear leukocytes. The WG was limited to the lung in seven patients. Fourteen patients in this series died because of active disease and two patients died because of iatrogenic complications. An improved knowledge of clinical and imaging features of WG could help the clinician reach an earlier diagnosis.

Relationship between Milk Intake and Mucus Production in Adult Volunteers Challenged with Rhinovirus-2—CB Pinnock, NM Graham, A Mylvaganam, RM Douglas. Am Rev Respir Dis 1990; 141:352.

In the first of three studies investigating the widely held belief that 'milk produces mucus,' 60 volunteers were challenged with rhinovirus-2, and daily respiratory symptoms and milk and dairy product intake records were kept over a 10-day period. Nasal secretion weights were obtained by weighing tissues collected and sealed immediately after use. Information was obtained on 51 subjects, yielding 510 person-days of observation. Subjects consumed zero to 11 glasses of milk per day (mean, 2.7; SE, 0.08), and secretion weights ranged from zero to 30.4 g/day (mean, 1.1; SE, 0.1). In response to an initial questionnaire, 27.5% reported the practice of reducing intake of milk or dairy products with a cold or named milk or dairy products as bad for colds. Of the latter group, 80% stated the reason as "producing more mucus/phlegm." Milk and dairy product intake was not associated with an increase in upper or lower respiratory tract symtoms of congestion or nasal secretion weight. A trend was observed for cough, when present, to be loose with increasing milk and dairy product intake; however, this effect was not statistically significant at the 5% level. Those who believe 'milk makes mucus' or reduce milk intake with colds reported significantly more cough and congestion symptoms, but they did not produce higher levels of nasal secretions. We conclude that no statistically significant overall association can be detected between milk and dairy product intake and symptoms of mucus production in healthy adults, either asymptomatic or symptomatic, with rhinovirus infection.

Dose-Response Effects of Two Sizes of Monodisperse Isoproterenol in Mild Asthma—P Patel, D Mukai, AF Wilson. Am Rev Respir Dis 1990; 141:357.

The dose-response effect of monodispersed isoproterenol of two different sizes (diameters 2.5 and 5 μm) was examined in eight mild asthmatic subjects (baseline FEV_1 81.5% of

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predicted). Pulmonary and cardiovascular variables were measured before and following 1, 2, 4, 8, and 16 cumulative min of aerosol inhalation. Subjects inhaled 1 to 30 μg (2.5-μm particles) or 2 to 50 μg (5-μm particles) of isoproterenol. Pulmonary but not cardiac responses were significantly greater for the 2.5-μm particles as compared to equivalent doses of 5-μm particles. Pulmonary dose-related response differences were particularly marked for variables associated with small airway function (FEF_{25-75} and FEF_{75-85}). These findings suggest that small particles penetrate more deeply into the lung and thereby more effectively dilate small airways and that small amounts of appropriately sized inhaled bronchodilator may produce considerable therapeutic effects.

Pressurized Aerosol versus Jet Aerosol Delivery to Mechanically Ventilated Patients—HD Fuller, MB Dolovich, G Posmituck, WW Pack, and MT Newhouse. Am Rev Respir Dis 1990; 141:440.

The purpose of this study was to compare desposition of aerosol to the lung from a metered-dose inhaler (MDI) and aerosol holding chamber and from a jet nebulizer in ventilator-dependent patients. Twenty-one patients were entered into the study, all receiving assisted ventilation and inhaled bronchodilators because of airflow limitation. The average age was 68 yr; there were 10 men and 11 women. The patients were randomized to receive either 4 puffs (800 μg) of radiolabeled fenoterol by MDI of 1.75 mL (1,750 μg) of radiolabeled fenoterol solution by nebulizer. Imaging of lung fields was made by a portable scintillation camera at 5-min intervals during the study. Results showed that 20 patients completed the study, 9 receiving fenoterol by MDI, and 11 by jet nebulizer. Four were excluded from analysis because of previous pneumonectomy, two from each group. Lung deposition measured as a percent of given dose from either system was 5.65 $\pm$ 1.09 (mean SEM) for MDI plus extension chamber and 1.22 $\pm$ 0.35 for jet nebulizer ($p < 0.001$). Therefore, this trial shows significantly greater efficiency of aerosol deposition to the lung in ventilator-dependent patients when using an MDI plus aerosol holding chamber than when using a jet nebulizer.

Hemodynamic Effects of Negative-Pressure Ventilation in Patients with COPD—N Ambrosino, F Cobelli, A Torbicki, et al. Chest 1990;97:850.

In order to evaluate the hemodynamic effects of intermittent negative-pressure ventilation (INPV), eight patients with COPD (FEV_1/FVC, 54 $\pm$ 6%; mean $\pm$ SD), respiratory failure (P_{aO_2}, 52 $\pm$ 6 torr; P_{aCO_2}, 56 $\pm$ 4 torr), and clinical signs of inspiratory muscle fatigue underwent right cardiac catheterization while performing 20 minutes of INPV by a cuirass ventilator at a pressure (–20 to –40 cm H_2O) able to reduce the diaphragmatic electromyographic activity. Patients showed a mild basal pulmonary artery hypertension. During INPV, no changes in the mean values of HR (from 79 $\pm$ 20 to 80 $\pm$ 18 beats per minute), systolic BP (141 $\pm$ 19 to 139 $\pm$ 16 torr), cardiac output (5.2 $\pm$ 0.8 to 5.1 $\pm$ 1.3 L/min), mean pulmonary artery pressure (23.8 $\pm$ 3.8 to 23.9 $\pm$ 4.4 torr), right atrial pressure (4.3 $\pm$ 2.6 to 5.5 $\pm$ 2.5 torr), pulmonary wedge pressure (10.3 $\pm$ 4.5 to 9.4 $\pm$ 2.9 torr), total pulmonary resistance (369 $\pm$ 76 to 392 $\pm$ 124 dynes $\cdot$ s $\cdot$ cm^{-5}), and pulmonary vascular resistance (199 $\pm$ 51 to 233 $\pm$ 94 dynes $\cdot$ s $\cdot$ cm^{-5}) were observed. Direct systemic BP monitoring could be performed in six patients. During INPV, three patients showed 'pulsus paradoxus,' as assessed by an inspiratory fall in systolic BP of 11, 13, and 20 torr, respectively. We conclude that INPV by cuirass ventilator does not induce adverse hemodynamic effects in patients with COPD who have pulmonary artery hypertension.

Changes in the Contribution of the Rib Cage to Tidal Breathing during Infancy—MB Hershenson, AA Colin, MEB Wohl, AR Stark. Am Rev Respir Dis 1990;141:922.

As the shape, compliance, and deformability of the rib cage (RC) change during infancy, RC participation in quiet breathing may increase. We used respiratory inductive plethysmography (RIP) to determine the relative contributions of the RC and abdomen (AB) to tidal volume (V_T) in 20 studies in 14 healthy infants 1 to 26 months of age during quiet natural sleep. RIP was calibrated with simultaneous flow measurements (anesthesia mask and pneumotachograph) by the least squares method of statistical analysis. We analyzed segments of breathing with and without flow measurement for RIP-derived V_T, change in RC volume (V_{rc}) and AB volume (V_{ab}) with each breath and the RC contribution to tidal breathing (%RC = $V_{rc}/[V_{rc} + V_{ab}]$). The %RC increased with age: %RC = 1.4 age (months) + 33 ($r = 0.69$, $p < 0.01$). After 9 months of age, %RC resembled that found in quietly sleeping adolescents. Mask placement increased V_T in all but one subject (mean increase, 29 $\pm$ 23% of baseline $\pm$ SD; $p < 0.001$, paired t test). In infants younger than 10 months of age, mask placement also increased %RC (without mask, 40 $\pm$ 9%; with mask, 46 $\pm$ 10%; $p < 0.02$). We conclude that by 1 yr of age, the RC contribution to tidal breathing during quiet sleep is similar to that of the adolescent, suggesting that major developmental changes in RC shape, compliance, and deformability take place during infancy.

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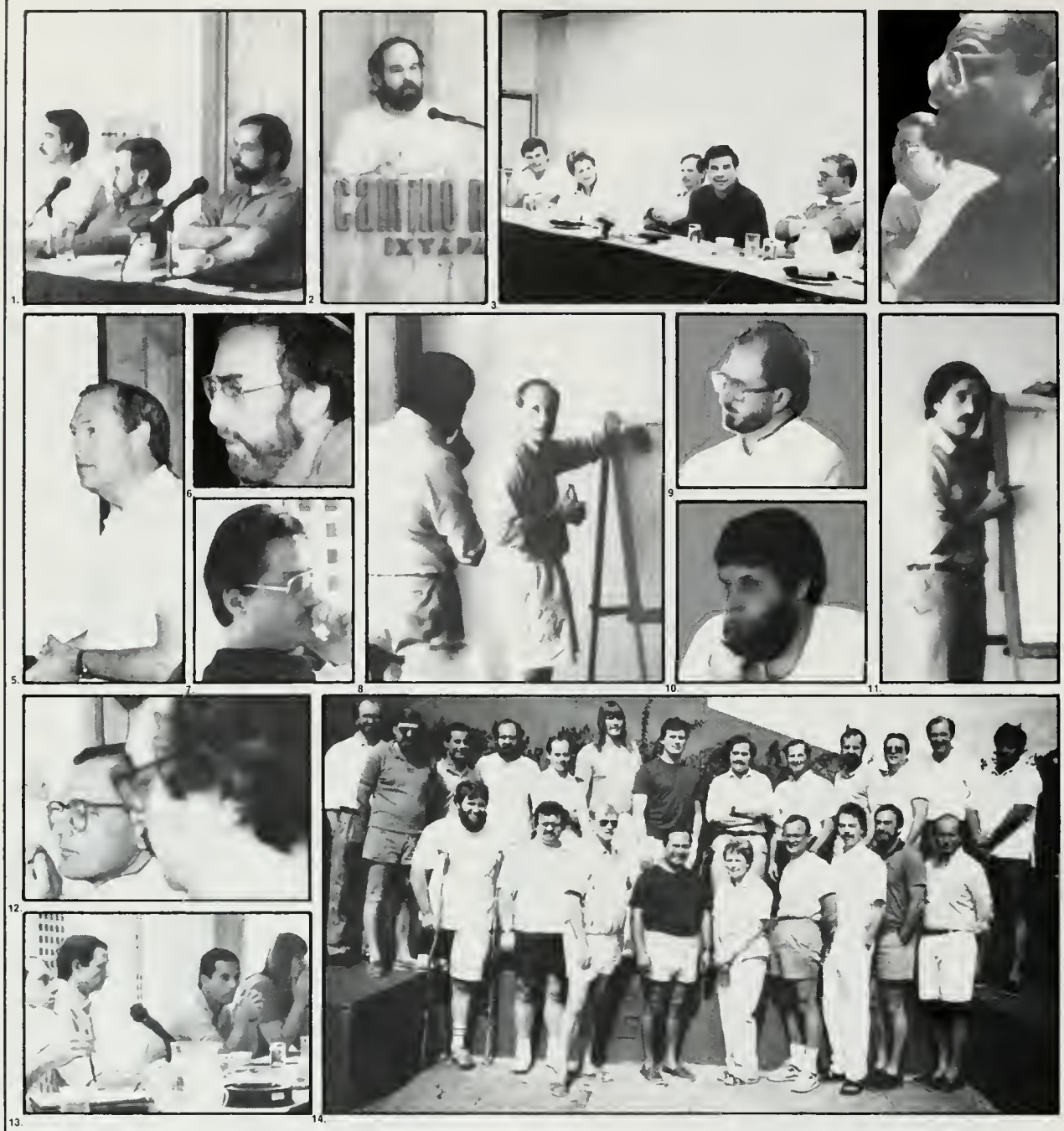

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Although the need to feed patients seems intuitively obvious, the importance of nutrition and its multiple complex interactions with numerous organ systems has only recently been appreciated. Similarly, total parenteral nutrition (TPN) is a comparatively new medical therapy, having been introduced by Dudrick and co-workers in 1969.[1] The intensive study of nutrition and metabolism has rekindled interest in the measurement of energy expenditure. The process of measuring energy expenditure is known as calorimetry and may be direct or indirect; the instruments are known as calorimeters. I will discuss methods of energy measurement, the practical aspects of measurement, and clinical use of the information provided.

DIRECT CALORIMETRY

Direct calorimetry is the actual measurement of a subject's heat production. Direct calorimetry requires that the subject be confined to a small room or chamber for extended periods of time in order for reliable data to be obtained. Additionally, no caregivers may enter the room, the patient must remain at rest, and other sources of heat production must be eliminated (eg, compressors and motors). Direct calorimeters are very expensive and because of the requirements listed are impractical for use with sick patients. However, direct calorimetry has been invaluable in the agriculture industry where the study of animal metabolism and nutrition has led to increased production of meat, eggs, and milk at lowest possible cost.

INDIRECT CALORIMETRY

Indirect calorimetry is the calculation of heat production through the measurement of pulmonary gas exchange, specifically the measurement of oxygen consumption ($\dot{V}_{O_2}$) and carbon dioxide production ($\dot{V}_{CO_2}$). The measurements of $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ are converted to energy expenditure (Kcal/day) by application of the Weir equation:[2]

$$\text{Energy expenditure} = [(\dot{V}_{O_2})(3.941) + (\dot{V}_{CO_2})(1.11)]1440, \quad (1)$$

where $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ are expressed in L/min, and 1440 is equal to the number of minutes in a day. Another important determination obtained from indirect calorimetry is the respiratory quotient (RQ), obtained by dividing $\dot{V}_{CO_2}$ by $\dot{V}_{O_2}$ (ie, $\dot{V}_{CO_2}/\dot{V}_{O_2}$). The RQ can serve as an indicator of quality control because in humans it resides in a fairly narrow range (0.67-1.3), and is also used to determine substrate utilization.

Indirect calorimeters can be classified as one of two types, based on the method of $\dot{V}_{O_2}$ measurement. With open-circuit indirect calorimeters $\dot{V}_{O_2}$ is determined by measuring the difference between inspired and expired gas concentrations and minute ventilation ($\dot{V}_E$). With closed-circuit calorimeters $\dot{V}_{O_2}$ is determined by measuring the volumetric change from a reservoir of oxygen over time. Within these

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two main types, there are variations on the theme that make these systems different enough to be considered separately. Open-circuit calorimeters use either mixing-chamber, dilution, or breath-by-breath technology; and closed-circuit systems use the volumetric loss or replenishment techniques. Commercially available indirect calorimeters and their suppliers are shown in Table 1.

Table 1. Commercially Available Indirect Calorimeters

Mixing-Chamber Systems
Meta Scope, Cybermedic Inc, Louisville, Colorado
2900 System, SensorMedics Corp, Yorba Linda, California
MMC Horizon, SensorMedics Corp, Anaheim CA
Metabolic Gas Monitor 2, Utah Medical Products, Midvale, Utah
Engstrom Metabolic Computer, Gambro-Engstrom, Bromma, Sweden
Breath-by-Breath Systems
CCM, Medical Graphics Corp, St Paul, Minnesota
RAS (Respiratory Assessment Systems), Biochem International Inc, Waukesha, Wisconsin
4400 System, SensorMedics Corp, Yorba Linda, California
Dilution Systems
Deltatrac, SensorMedics Corp, Yorba Linda, California
Closed-Circuit Volume-Loss Systems
MRM-6000, Waters Instruments Inc, Rochester, Minnesota
Closed-Circuit Replenishment Systems
VVR-Q, Vital Signs, Totowa, New Jersey

Abbreviations Used in this Paper

F_{ECO_2}	— Fractional expired concentration of carbon dioxide
$F\bar{E}CO_2$	— Fractional mixed expired concentration of carbon dioxide
F_{EN_2}	— Fractional expired concentration of nitrogen
F_{EO_2}	— Fractional expired concentration of oxygen
$F\bar{E}O_2$	— Fractional mixed expired concentration of oxygen
F_{ICO_2}	— Fractional inspired concentration of carbon dioxide
F_{IO_2}	— Fractional inspired concentration of oxygen
FRC	— Functional residual capacity
IMV	— Intermittent mandatory ventilation
N_2	— Nitrogen
OCB	— Oxygen cost of breathing
PEEP	— Positive end-expiratory pressure
Q	— Total flow
REE	— Resting energy expenditure
RQ	— Respiratory Quotient
TPN	— Total parenteral nutrition
$\dot{V}_{CO_2}$	— Carbon dioxide production
$\dot{V}_E$	— Expired minute ventilation
$\dot{V}_I$	— Inspired minute ventilation
$\dot{V}_{O_2}$	— Oxygen consumption
V_T	— Tidal volume

Open-Circuit Calorimetry

Theory and Calculations

Both the mixing-chamber and breath-by-breath variations of the open-circuit technique utilize the same measurements and formulas to calculate $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$. The required measurements are inspired and expired concentrations of oxygen (F_{IO_2}, F_{EO_2}) carbon dioxide (F_{ICO_2}, F_{ECO_2}) and inspired and expired minute ventilation ($\dot{V}_I$, $\dot{V}_E$). Temperature and pressure must also be measured because of their effect on gases. In an ideal situation, $\dot{V}_{O_2}$ would be calculated by the equation:

$$\dot{V}_{O_2} = \dot{V}_I (F_{IO_2}) - \dot{V}_E (F_{EO_2}), \quad (2)$$

and $\dot{V}_{CO_2}$ by a similar equation:

$$\dot{V}_{CO_2} = \dot{V}_E (F_{ECO_2}) - \dot{V}_I (F_{ICO_2}). \quad (3)$$

However, because it is technically difficult to measure the small differences between $\dot{V}_I$ and $\dot{V}_E$, $\dot{V}_I$ is commonly calculated using the Haldane transformation (Equation 10), which assumes that the relatively insoluble gas nitrogen (N_2) is constant in both inspired and expired gases. If there is no net nitrogen uptake, then $\dot{V}_I$ can be calculated by the equation:

$$\dot{V}_I = \frac{F_{EN_2}}{F_{IN_2}} \dot{V}_E, \quad (4)$$

where F_{EN_2} is the expired nitrogen concentration and F_{IN_2} is inspired nitrogen concentration. If there are no gases present other than N_2, O_2, and CO_2 (eg, anesthetic agents), F_{EN_2} and F_{IN_2} can be determined from measuring O_2 and CO_2 alone:

$$F_{IN_2} = 1 - F_{IO_2} - F_{ICO_2}, \quad (5)$$

and

$$F_{EN_2} = 1 - F_{EO_2} - F_{ECO_2}. \quad (6)$$

Substituting these calculations into Equation 2, the formula for $\dot{V}_{O_2}$ becomes:

$$\dot{V}_{O_2} = \frac{(1 - F_{EO_2} - F_{ECO_2})}{1 - F_{IO_2}} (F_{IO_2} - F_{EO_2}) \dot{V}_E. \quad (7)$$

This equation is used by many commercially available systems, although a few actually do measure $\dot{V}_I$. Failure to account for these differences, and assuming that $\dot{V}_I$ equals $\dot{V}_E$, can lead to clinically significant errors in $\dot{V}_{O_2}$ measurement. For example, a patient breathing 10 times per minute at a 1000-mL tidal volume (V_T) who has a $\dot{V}_{O_2}$ of 250 mL/min and an RQ of 0.7 will consume 25 mL of O_2 per breath while producing 17.5 mL of CO_2 per breath. In this case, the difference between $\dot{V}_I$ and $\dot{V}_E$ is 75 mL. If $\dot{V}_{O_2}$ were calculated assuming $\dot{V}_I$ equals $\dot{V}_E$, $\dot{V}_{O_2}$ would be reported erroneously as 208 mL/min—an error of 17%.[3] As RQ approaches 1.0, this error becomes smaller. $\dot{V}_{CO_2}$ is a much simpler measurement; in mechanically ventilated patients and subjects breathing through a mask or mouthpiece, the equation for $\dot{V}_{CO_2}$ can be simplified to:

$$\dot{V}_{CO_2} = \dot{V}_E (F_{ECO_2}). \quad (8)$$

This is possible because during mechanical ventilation F_{ICO_2} is 0.0% and room air contains only minute amounts of CO_2, usually less than 0.03%. However, when a flow-by system is used, F_{ICO_2} must be accounted for. The key components of all open-circuit calorimeters are the analyzers (O_2 and CO_2) and a volume-measuring device. The analyzers must be capable of measuring small changes in gas concentrations (0.001%) in room air and oxygen-enriched environments. The volume-measuring device may be a turbine spirometer or pneumotachometer, and must be capable of accurately measuring volumes from 0.01 to 2.0 L.

Another important component is the pressure transducer. Because most O_2 analyzers in indirect calorimeters measure the partial pressure of oxygen, changes in pressure (such as those that occur during mechanical ventilation) will affect the measurement of F_{IO_2}. The pressure transducer corrects the analyzers for periodic changes in total pressure.

Mixing-Chamber Technique

The majority of commercially available indirect calorimeters use mixing chambers. Such systems can be used to perform measurements on spontaneously breathing patients via a canopy, mouthpiece, or mask and on those receiving mechanical ventilation. A generic open-circuit system is shown in Figure 1. Expired gas from the patient is directed into the mixing chamber where baffles interrupt flow and prevent streaming of gases and uneven gas concentrations. At the end of the mixing chamber, a vacuum pump withdraws a small sample of mixed expired gas for measurement of $F_{\bar{E}O_2}$ and $F_{\bar{E}CO_2}$ by the O_2 and CO_2 analyzers. A pressure transducer is part of this circuit to assure pressure-compensated gas measurements. This sample of gas is returned to the mixing chamber after analysis. At preselected intervals, the analyzers also measure the inspired gas concentration for determination of inspired and expired differences; the entire volume of gas then exits through a volume transducer for measurement of minute ventilation. A thermistor is used for temperature correction of volumes. A microprocessor system controls the calorimeter functions, writes the data to the memory, and performs the necessary calculations. A printed copy of the information is usually available.

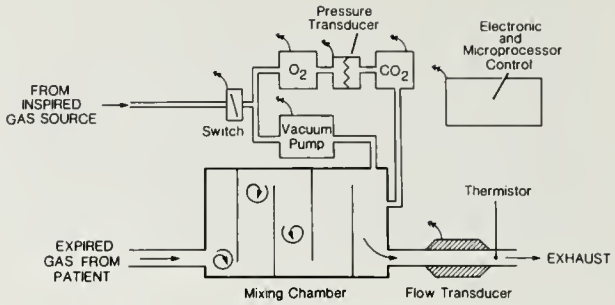

Fig. 1. Diagram of an open-circuit calorimeter incorporating a mixing chamber.

Modification of the mixing-chamber system allows measurements to be made in a canopy. Sometimes referred to as a flow-through or flow-by system, this system allows the patient to be measured unencumbered by a mask, mouthpiece, or nose clips. These measurements are accomplished by placing the patient inside a plastic hood that has two openings. One opening is connected by large-bore tubing to a blower or fan that draws room air through the second opening. This gas flows over the patient's face and with the patient's own expired gases travels to the mixing chamber (Fig. 2). The process of $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ measurement is the same. Because the patient's

expired gases are diluted by room air, the difference in inspired and expired gas concentrations is very small. This mandates that the analyzers be precise and free of drift. Control of flow through the canopy is also important because too low a flow will cause CO_2 to rise in the canopy stimulating respiration and too high a flow will cause excessive dilution of expired gases.

Breath-by-Breath Technique

The open-circuit breath-by-breath technology differs from the mixing-chamber system in that there is no mixing chamber, measurements are made every breath, and the site of gas sampling and volume measurements is usually at the airway. Figure 3 (top) depicts a typical breath-by-breath system. Gas samples for measurement of F_{IO_2}, F_{EO_2}, F_{ICO_2}, and F_{ECO_2} are analyzed for every breath as is the tidal volume. Because there is no mixing chamber, the measurements are arithmetically averaged at a clinician-selected or factory preset time to obtain mixed expired values. In some systems, the volume-measurement device is moved from the airway to a more distal location to avoid errors in measurement caused by humidity and secretions. Breath-by-breath systems were initially designed for exercise testing, but have been modified to make them adaptable to patients on mechanical ventilation and spontaneously breathing patients using a mask or mouthpiece or, more recently, a canopy device. The canopy

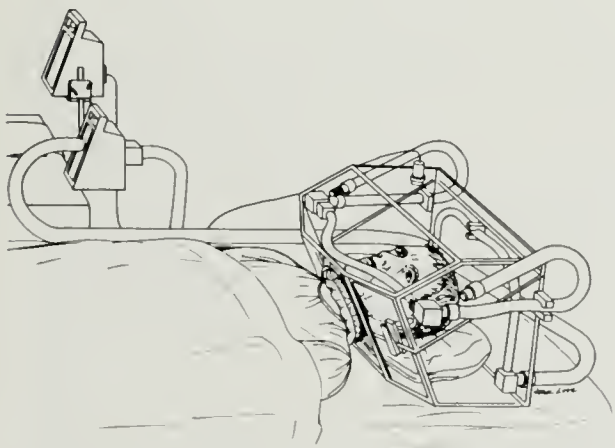

Fig. 2. Artist's rendering of a canopy system used in open-circuit calorimeters.

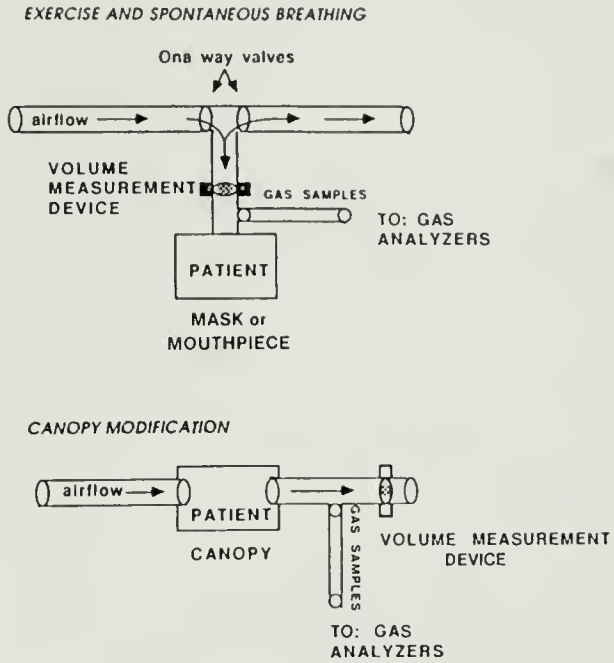

Fig. 3. Placement of gas sampling lines and volume-measurement device for breath-by-breath open-circuit technique (top). Modification of the breath-by-breath system to allow canopy measurements (bottom). (Reprinted, with permission, from Reference 16.)

modification is shown in Figure 3 (bottom). Samples are taken on a breath-by-breath basis and averaged to obtain mixed expired values.

Dilution Technique

The dilution principle is a modification of the open-circuit technique, and, therefore, the instrumentation is similar (Fig. 4). Only the SensorMedics 2900 system and Deltatrac currently use the dilution principle. The Deltatrac consists of a 4-L mixing chamber, a paramagnetic oxygen analyzer, infrared CO_2 analyzer, microprocessor, and CRT display. A precise flow-generating system that dilutes expired gases with room air replaces the volume-measuring device and gives the principle its name.

During measurement of mechanically ventilated patients, expired gas travels to the mixing chamber where mixed expired gases ($F_{\bar{E}O_2}$, $F_{\bar{E}CO_2}$) are measured. After passing through the mixing chamber, the gases are diluted with room air by the flow generator so that total flow (Q) through the system

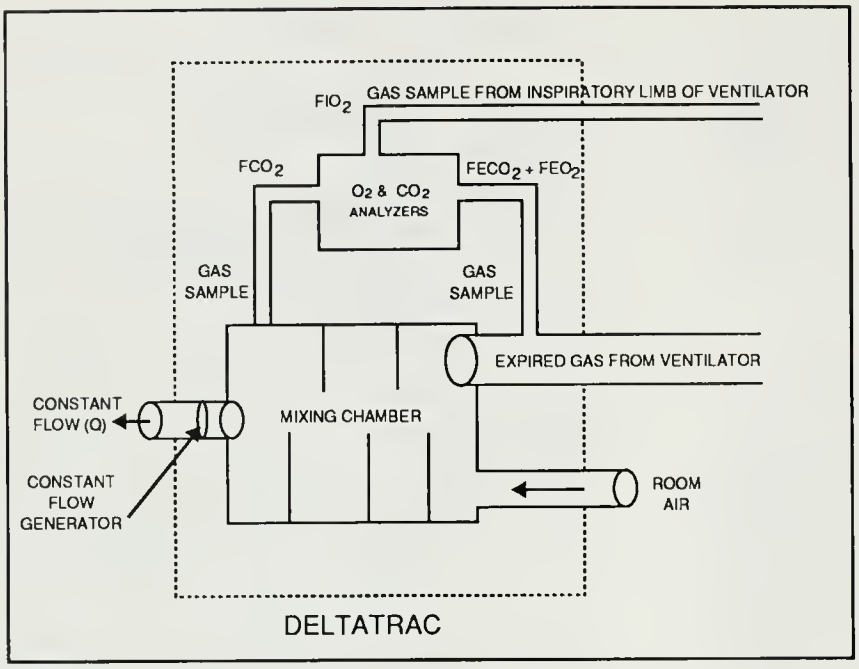

Fig. 4. Schematic of the dilution system utilized by the Deltatrac during mechanical ventilation. (Reprinted, with permission, from Reference 4.)

is equal to the flow generator's output (usually 40 L/min). $\dot{V}_{CO_2}$ is then calculated as:

$$\dot{V}_{CO_2} = (Q)(F_{E^*CO_2}), \quad (9)$$

where $F_{E^*CO_2}$ represents the concentration of CO_2 in the diluted gas sample. After $\dot{V}_{CO_2}$ has been determined, RQ is calculated by the Haldane transformation:

$$RQ = \frac{1 - F_{IO_2}}{\frac{F_{IO_2} - F_{EO_2}}{F_{ECO_2}} - F_{IO_2}}. \quad (10)$$

Oxygen consumption is then determined by the formula:

$$\dot{V}_{O_2} = \frac{\dot{V}_{CO_2}}{RQ}. \quad (11)$$

Gas sampling is controlled by the microprocessor. Measurements of CO_2 occur by alternating sampling between F_{ECO_2} and $F_{E^*CO_2}$. Measurements of O_2 occur by alternating between F_{IO_2} and the F_{IO_2}-$F_{\bar{E}O_2}$ difference.

During canopy measurements, the determinations of $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ are altered. In this instance, the constant-flow generator serves to draw gases through the canopy and into the mixing chamber (Fig. 5). $\dot{V}_{CO_2}$ is calculated as:

$$\dot{V}_{CO_2} = Q(F_{CO_2}). \quad (12)$$

$\dot{V}_{O_2}$ is determined by:

$$\dot{V}_{O_2} = \frac{Q}{1 - F_{IO_2}}[F_{O_2} - (F_{IO_2})(F_{CO_2})]. \quad (13)$$

RQ can then be determined as previously described:

$$RQ = \frac{\dot{V}_{CO_2}}{\dot{V}_{O_2}}. \quad (14)$$

The accuracy of this device utilizing the dilution principle has been reported by Weissman et al[4] as $\pm$ 7% and by Takala et al[5] as $\pm$ 4%.

Technical Considerations

Indirect calorimeters are generally easy to understand and operate. However, in the clinical arena, many variables must be taken into account to allow accurate measurement—the stability and absolute level of F_{IO_2}, the separation of inspired and expired gases, complete collection of expired gases, and elimination of water vapor. In addition, the individual components of the system (eg, gas analyzers and flow-measuring devices) must function appropriately, be calibrated frequently, and be tested against known standards.

Effects of F_{IO_2}. Ultman and Bursztein[6] and Halmagyi and Kinney[7] have described in detail the untoward effects of elevated F_{IO_2} on open-circuit $\dot{V}_{O_2}$

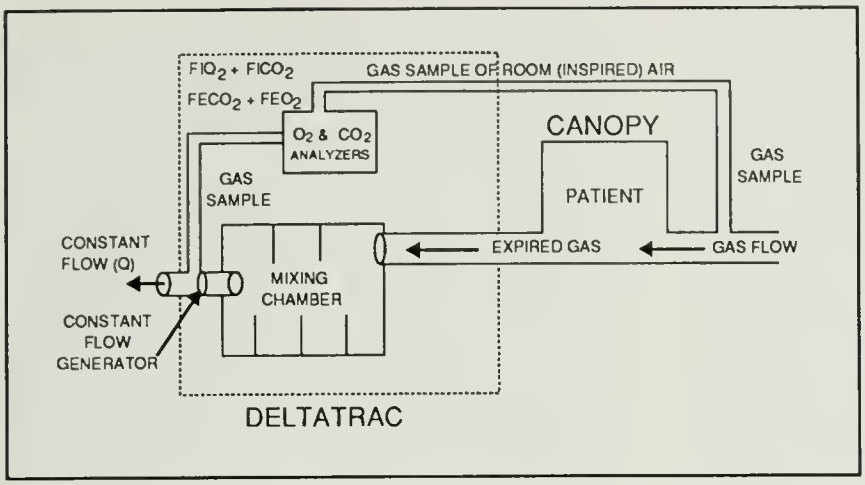

Fig. 5. Diagram of the dilution system utilized by the Deltatrac during canopy measurements. (Reprinted, with permission, from Reference 4.)

measurements. Error is introduced when F_{IO_2} approaches 1.0 and the denominator of the Haldane equation ($1 - F_{IO_2}$) becomes minute or zero. (Division by zero yields infinity.) Additionally, any error in measurement of gas concentrations is amplified as F_{IO_2} increases. An error in F_{IO_2} measurement of 1% at an F_{IO_2} of 0.40 results in an error in $\dot{V}_{O_2}$ measurement of 15%. At an F_{IO_2} of 0.80 or more, the same 1% error results in an error almost equal to the true $\dot{V}_{O_2}$ value.[4] Several solutions to this problem have been suggested—accurately measuring $\dot{V}_I$ with a volume-monitoring device at the airway,[8] improving O_2 sensor design,[9] and increasing the frequency at which samples are analyzed for gas concentrations.[6]

Improvement in O_2-sensor design seems to be the most practical and has often been attempted. Early open-circuit indirect calorimeters were validated and found to be accurate up to an F_{IO_2} of 0.40.[10] More recent equipment has been found to be accurate up to 0.80.[5,6] Much of this improvement can be attributed to improved sensor design.

During measurements at elevated oxygen concentrations, the stability of the oxygen concentration is also critically important. Browning et al[11] have demonstrated the deleterious effects of fluctuating F_{IO_2} during metabolic measurements at elevated oxygen concentrations. They noted that during mechanical ventilation, F_{IO_2} fluctuated with the delivery of a ventilator breath. Because most systems only measure F_{IO_2} periodically, if F_{IO_2} changes between F_{IO_2} analysis and expired-gas collection, the $\dot{V}_{O_2}$ measurement will be incorrect. If the actual F_{IO_2} increases after F_{IO_2} analysis, measured $\dot{V}_{O_2}$ will be an underestimation of actual $\dot{V}_{O_2}$. If F_{IO_2} falls after F_{IO_2} analysis, measured $\dot{V}_{O_2}$ will be an overestimation of actual $\dot{V}_{O_2}$. Browning and colleagues suggested using an air-oxygen blender between the wall gas source and ventilator to alleviate this problem. The use of the air-oxygen blender with mechanical ventilators and during mouthpiece or mask measurements at increased F_{IO_2} should be considered mandatory. Some third-generation ventilators that utilize external blenders and/or a reservoir prior to delivery of gas into the patient circuit may not require use of a second blender. Internal reservoirs (such as those found on the Hamilton Veolar, Hamilton Medical, Reno NV; Siemens 900C, Siemens-Elema Ventilator Systems, Schaumburg IL; Ohmeda-Advent, Ohmeda, Louisville CO; and Bird 6400ST, Bird Products Corp, Palm Springs CA) serve as 'inspiratory mixing chambers,' damping any fluctuations in F_{IO_2}. Ventilators that use on-line mixing of air and O_2 (eg, the proportional valves of the Puritan-Bennett 7200a) are prone to frequent changes in F_{IO_2} as patient demand changes. In my experience, as $\dot{V}_E$ increases, the stability of the F_{IO_2} of many ventilators is adversely affected.

Breath-by-breath systems that measure F_{IO_2} during every inspiration may help to minimize changes caused by fluctuating F_{IO_2}. However, although this is theoretically possible, to my knowledge it has not been systematically studied. Frequent sampling of F_{IO_2} also helps to minimize errors caused by periodic fluctuations. Generally, this requires that F_{IO_2} be sampled at least once per minute.

Effects of Anesthetic Gases. The presence of anesthetic gases invalidates open-circuit technique accuracy. Error is introduced when the Haldane equation is used when gases other than N_2, O_2, and CO_2 are present.[12,13]

Separation of Inspired and Expired Gas. Separation of inspired and expired gases is also of paramount importance. This is particularly true in systems with any type of continuous flow. If continuous flow is allowed to enter the mixing chamber along with expired gases, measured $\dot{V}_E$ will be drastically increased, and gas concentrations will be diluted. Nonrebreathing valves or double-piloted, or dual, exhalation valves placed at the airway will help to solve this problem.[14,15] Caution should be used to assure that any valve system placed between the patient and ventilator does not adversely affect the work of breathing. During mechanical ventilation with control, assist-control, pressure support, IMV, or CPAP with a demand valve, expired gases can simply be collected from the expiratory port of the ventilator. Although the compressible volume of the circuit will cause the calorimeter to overestimate $\dot{V}_E$, dilution of expired gases by the same volume negates any change.[16]

System Leaks. During measurement, the ventilator circuit and tubing to the calorimeter must be leak free. Any loss of gas to the environment or any entrainment of room air will result in erroneous data. All expired gases must also be collected. Patients with incompetent endotracheal tube cuffs, leaking chest tubes, or bronchopleural fistulas will lose expired gases to the environment. This results in decreased $\dot{V}_E$ and $F\bar{E}CO_2$ and leads to underestimation of $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and resting energy expenditure (REE).[17] In this instance, the RQ may be correct, but this is of little value when other variables are inaccurate.

Effects of Water Vapor. Water vapor adversely affects the function of O_2 and CO_2 analyzers and volume-monitoring devices.[18] Water vapor should be eliminated from gas prior to its reaching the analyzers to ensure accurate measurements. Elimination of water vapor can be accomplished by desiccants,[19] superheating of the gas sample,[20] cooling of the gas sample,[21] water traps,[22] or tubing that allows equalization of water vapor in the sampling line with that of ambient air.[22] Desiccants were initially used, but they can delay the transit of gases and increase the cost per test. Elimination of water vapor through heating, cooling, or specialized tubing appears to be the most efficient and cost-effective approach.

Adequate Sensors. Appropriate sensors that are capable of responding quickly to changes in gas concentrations but that also maintain reproducibility and stability are required. Current open-circuit calorimeters use either a zirconium or paramagnetic O_2 sensor. The zirconium-oxide cell possesses a fast response time and is highly accurate; however, a disadvantage of this type of O_2 cell is that it must be heated to temperatures near 800 °C.[23] Paramagnetic cells are also quick to respond to changes in F_{IO_2} and maintain excellent stability. Recently, a differential-pressure paramagnetic cell has been introduced that measures the difference between two gases, rather than the absolute value of either.[9] This type of O_2 cell is ideally suited to metabolic measurements. CO_2 analyzers are generally infrared sensors with excellent stability and reliability. Volume-monitoring devices should be linear across a wide range of flows. Errors in measurement at either the low or high end of the volume range may make the device unsuitable for use in pediatrics and at high $\dot{V}_E$.

Calibration. Two-point calibration of the O_2 and CO_2 analyzers should be done daily and recorded. The initial calibration may be done with room air or 100% nitrogen to establish zero. The span or high point of the calibration should be relevant to the gas concentration to be measured clinically. That is, if a patient is receiving an F_{IO_2} of 0.35, then the calibration gas should have an $F_{IO_2} \geq 0.35$. This prevents errors caused by alinear sensors. Volume-monitoring devices should also be calibrated daily at clinically relevant volumes. Temperature and pressure must also be monitored to allow correction of gas concentrations and volumes according to the gas laws.

Measurement Validation. Validation of open-circuit calorimeters can be accomplished in two ways—by evaluating the RQ value or by measuring the $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$. Damask et al[24] have shown that burning methanol, which has a known RQ of 0.67, inside a lung model (Fig. 6), effectively tests the calorimeter's

ability to measure RQ. Several authors have described the addition of N_2 to simulate $\dot{V}_{O_2}$ and of CO_2 to simulate $\dot{V}_{CO_2}$ to a similar lung model (Fig. 7).[24-26] If N_2 and CO_2 are delivered via precision flowmeters, the calorimeter should reflect these measurements within 10%. Validation of an open-circuit calorimeter is time-consuming and can be dangerous when an open flame is burned in an oxygen-enriched environment. Appropriate precautions should be taken.

In general, validation should be undertaken to aid in troubleshooting a poorly functioning calorimeter and on a routine basis (every 4 to 6 months) as part of a quality assurance program.

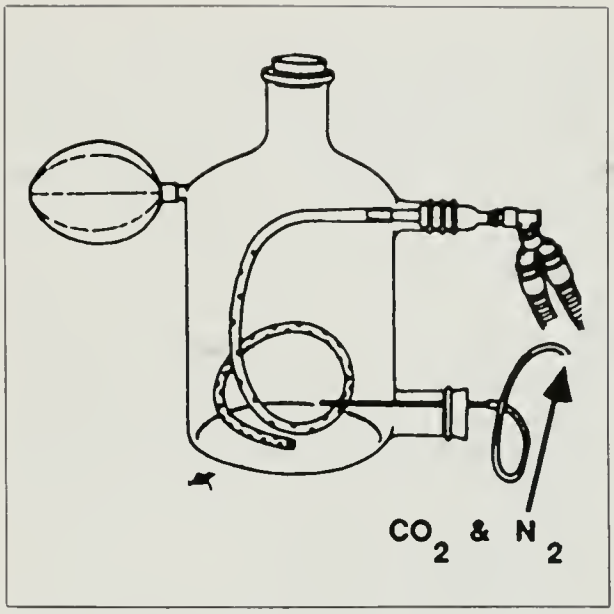

Fig. 7. Lung model for evaluation of $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$ measurements using N_2 dilution and CO_2 infusion.

Closed-Circuit Calorimetry

Volume-Loss Technique

Closed-circuit calorimetry using the volume-loss technique is often considered the 'gold standard'—primarily because the early measurements of energy expenditure, upon which the Harris-Benedict equation is based, were made using this technique.[27,28]

Early closed-circuit systems consisted of a water-seal spirometer filled with 100% oxygen, a CO_2 absorber, and a breathing circuit. The patient breathed to and from the spirometer via a mouthpiece. As O_2 was consumed and CO_2 absorbed by the soda lime, the volume in the spirometer decreased. The change in volume of the spirometer as a function of time allowed $\dot{V}_{O_2}$ to be measured (in mL/min).

At present, only the Waters Instruments MRM-6000 uses this technology. A generic closed-circuit system is shown in Figure 8. Closed-circuit systems allow measurements to be obtained during mechanical ventilation and during spontaneous ventilation via mask or mouthpiece and nose clip. Canopy measurements are not possible. During measurement, the patient inspires oxygen from the spirometer and exhales into the mixing chamber. If the patient requires mechanical ventilation, the ventilator

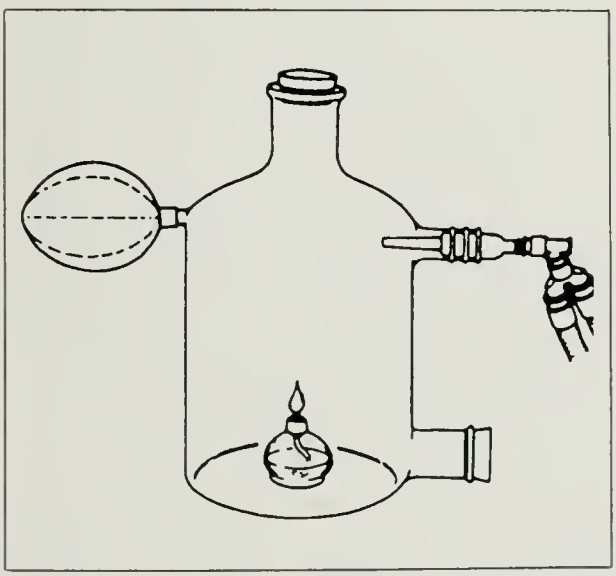

Fig. 6. Lung model for evaluating RQ measurements.

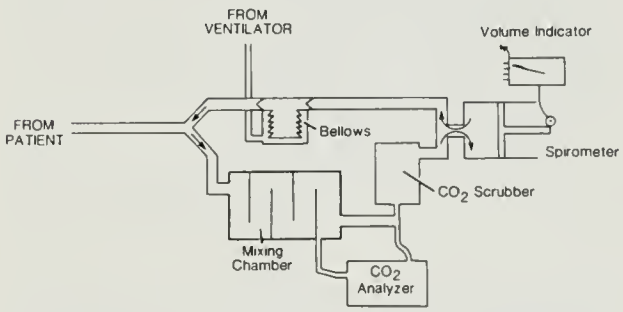

Fig. 8. Diagram of a generic closed-circuit calorimeter that utilizes the volume-loss technique.

pressures a canister that surrounds a bellows. This forces the bellows upward and delivers gas to the patient—in a manner similar to the bag-in-the-box technique of ventilation used during anesthesia. Gas entering the mixing chamber is analyzed for $F\bar{E}CO_2$ prior to passing through the soda lime CO_2 scrubber. The to-and-fro movement of the spirometer records V_T and $\dot{V}_E$. The MRM-6000 actually uses a turbine spirometer to measure V_T and $\dot{V}_E$. Change in the end-expiratory position of the spirometer is electronically measured and corrected for time, allowing determination of $\dot{V}_{O_2}$.[29] CO_2 is determined from measurements of volume and $F\bar{E}CO_2$ as previously described in the open-circuit system.

Volume-Replenishment Technique

Closed-circuit indirect calorimeters that use the volume-replenishment technique differ from their counterparts by maintaining the volume from the spirometer constant rather than assessing depletion. The Vital Signs VVR-Q system is presently the only commercially available device utilizing this technique. Figure 9 depicts the VVR-Q, which is essentially a

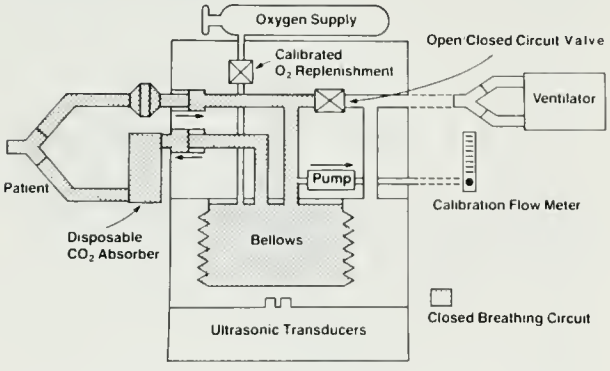

Fig. 9. Diagram of the Vital Signs VVR closed-circuit calorimeter, which incorporates the replenishment technique.

bag-in-a-box-system. Measurements can be made via mask, mouthpiece and nose clip, and during mechanical ventilation. Similar to the Waters MRM-6000, the VVR-Q consists of a CO_2 absorber, breathing circuit, and oxygen reservoir (in this case a bellows). This system monitors the movement of the bellows with ultrasonic transducers inside the canister to determine V_T and $\dot{V}_E$. $\dot{V}_{CO_2}$ is measured as previously described. With this instrument, the bellows serves the dual function of reservoir and mixing chamber. Measurement of $\dot{V}_{O_2}$ is depicted in Figure 10. The bellows is filled with oxygen, and its position is measured by an ultrasonic sensor (left). As the patient extracts O_2 and CO_2 is removed by an absorber, the position of the bellows changes (middle). This change in position is detected by the ultrasonic sensor. Calibrated pulses of O_2 from an external source are then fed into the bellows (right). The number and volume of the pulses necessary to return the bellows to its original position equal $\dot{V}_{O_2}$. Accuracy of the $\dot{V}_{O_2}$ measurement with the VVR-Q has been described by several authors and found to be excellent.[30–32]

Technical Considerations

Effects of F_{IO_2}. Closed-circuit calorimetry is unaffected by some of the problems that affect open-circuit calorimetry, is equally affected by others, and brings along a whole new set of its own problems. Because an O_2 analyzer is unnecessary, closed-circuit calorimetry eliminates difficulties associated with fluctuating F_{IO_2}. Use of the closed-circuit technique makes it possible to study patients who require an F_{IO_2} of 1.0. One difficulty with closed-circuit systems and F_{IO_2} occurs during measurement with room air. With the MRM-6000, F_{IO_2} diminishes as the test progresses. If the spirometer is filled with ambient air, the patient eventually receives an hypoxic gas mixture.[31] This problem does not appear to occur with the replenishment system.

Leaks. Prevention of leaks in the circuit is critical. Loss of volume from the circuit presents as a sharp rise in $\dot{V}_{O_2}$. Entrainment of room air into the circuit causes $\dot{V}_{O_2}$ to be underestimated. Fortunately, when either of these problems occurs, the magnitude of the leak is so great that $\dot{V}_{O_2}$ is wildly inaccurate. This should lead the operator to search for the problem. The VVR-Q utilizes a leak test prior to measurement to help reduce this problem.

Duration of Measurement Period. Duration of the measurement period using closed-circuit calorimetry is also of concern. With the MRM-6000, test time is limited by patient $\dot{V}_{O_2}$ and the volume of the

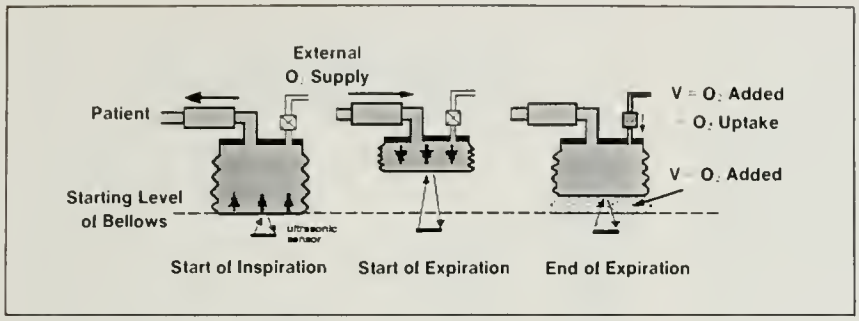

Fig. 10. Measurement of $\dot{V}_{O_2}$ by the VVR calorimeter.

spirometer. As $\dot{V}_{O_2}$ increases, measurement time decreases because the spirometer volume nears zero. The test can be interrupted and the spirometer refilled, but this is undesirable. Filling the spirometer with 100% oxygen increases the testing duration, but this procedure is not recommended. The effects of breathing 100% oxygen on oxygen consumption and lung function may alter accuracy of the measurement.[33] The replenishment technique solves the volume-depletion problem, but is limited by the life of the CO_2 absorber.[34] The VVR-Q provides CO_2 absorbers with a life of 1 hour. Homemade CO_2 canisters that are capable of holding a larger volume of soda lime, can help to prevent this problem.

Effects of FRC Changes. Both systems are rendered inaccurate by acute changes in end-expiratory lung volume. If functional residual capacity (FRC) falls during the course of the test, $\dot{V}_{O_2}$ will appear to decrease. If FRC rises, as might occur with air trapping, $\dot{V}_{O_2}$ will be overestimated.

Effects of Mechanical Ventilation. The most perplexing problems encountered with closed-circuit calorimetry occur during measurement of mechanically ventilated patients. Because the calorimeter is placed between the patient and the mechanical ventilator, patient-ventilator interaction is hampered. The internal volume of the calorimeter causes compressible volume to increase markedly. In order to assure the same V_T delivery, the ventilator V_T must be increased to overcome the compressible volume. The VVR-Q system provides a chart based on desired-volume delivery and peak pressure to assist in this calculation. I recommend that the actual volume delivered to the patient be measured prior to initiating the measurement. Patients with low lung compliance and high peak inspiratory pressure are a particular problem. In such a scenario, ventilator volume may be at its maximum level and the V_T delivered to the patient through the calorimeter inadequate. If this occurs, measurement should not be attempted. Positioning of the calorimeter between the patient and the ventilator also affects trigger sensitivity and the reproducibility of ventilator mode and prolongs inspiratory time and, hence, increases the work of breathing. Keppler et al[31] suggest that if patient work appears to increase, then the mechanical ventilator rate should be increased to compensate for the problem. Although this seems prudent, it violates an important rule of noninvasive monitoring—it changes the patient's condition simply for the sake of the measurement, which may render the data inapplicable to the patient's 'normal' condition. The MRM-6000 also will not faithfully reproduce pressure support ventilation or continuous-flow IMV. PEEP or CPAP using the MRM-6000 must be controlled by the calorimeter's own internal magnetic PEEP valve. This may also adversely affect the patient's work of breathing,[35] although this has not been studied. The VVR-Q system also changes patient-ventilator characteristics, which limits its applicability in many intensive care units, but it appears to more faithfully reproduce ventilator modes than does the MRM-6000.[31]

Open- vs Closed-Circuit Calorimeters

An awarenss of the difficulties and problems associated with each of the systems described should enable the reader to decide which system is appropriate for a particular application. A closed-circuit system is ideally suited for the patient on control or assist-control ventilation who requires a high F_{IO_2} (≥ 0.60). However, a closed-circuit system is poorly suited to the patient receiving low-rate IMV

(< 6 breaths/min) and PEEP who must provide a majority of $\dot{V}_E$ by spontaneous breathing.

Open-circuit systems are ideally suited for spontaneously breathing patients on room air (because of the canopy system) and for mechanically ventilated patients in any mode at an $F_{IO_2} < 0.60$. Closed-circuit systems may also be advantageous with the spontaneously breathing patient with an intact airway who requires supplemental oxygen. This is true because the reservoir can be filled with gas of the required O_2 concentration from which the patient can breathe. An open-circuit system in this instance requires an air-oxygen blender and a high-flow aerosol system with a series of T-pieces and one-way valves assembled prior to measurement to direct inspired and expired gas flow.

Comparative evaluations of open-circuit and closed-circuit systems are scant.[32,36] We recently compared $\dot{V}_{O_2}$ measurements made with the VVR (prior to its ability to measure $\dot{V}_{CO_2}$, the VVR-Q was simply VVR) to those made with the Deltatrac.[36] We chose three groups of patients under four conditions. The first two conditions utilized the same 10 volunteers. In Group 1, 10 volunteers had their $\dot{V}_{O_2}$ measured with the Deltatrac with the canopy system, then by the VVR with mouthpiece and nose clip, and again by the Deltatrac. Subjects in Group 2 were measured in the same sequence, but all measurements were made with a mouthpiece and nose clip. Group 3 consisted of patients who required mechanical ventilation in the assist-control mode. Group 4 consisted of patients who required mechanical ventilation with IMV and PEEP. In all instances, measurements were made until $\dot{V}_{O_2}$ was stable for at least 10 minutes. The sequence of measurements for Groups 3 and 4 remained Deltatrac-VVR-Deltatrac. We found an excellent correlation between $\dot{V}_{O_2}$ measurements made by the Deltatrac and VVR (Fig. 11) and between the two Deltatrac measurements (Fig. 12). Analysis of data by group demonstrated no statistically significant differences in the $\dot{V}_{O_2}$ measurements in Groups 1, 2, and 3. In Group 4, closed-circuit measurements averaged 16% greater than either open-circuit measurement (Fig. 13). Based on these data and our observation of the patients, we concluded this was due to the adverse effects on the patients' work of breathing. Group 1 volunteers breathing through the VVR with mouthpiece and nose clips also had a slightly higher $\dot{V}_{O_2}$

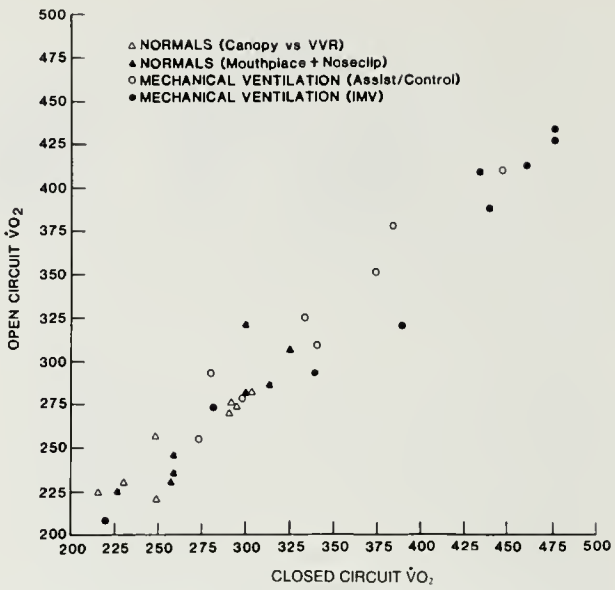

Fig. 11. Correlation of $\dot{V}_{O_2}$ measurements made by the Deltatrac and the VVR calorimeter. r = 0.974.

than when they were in the canopy. When mouthpiece and nose clips were used for both open-circuit and closed-circuit measurements, this difference was not observed. We believe the differences in Group 1 were due to the effects of breathing through a mouthpiece while wearing a nose clip. Adverse effects of the

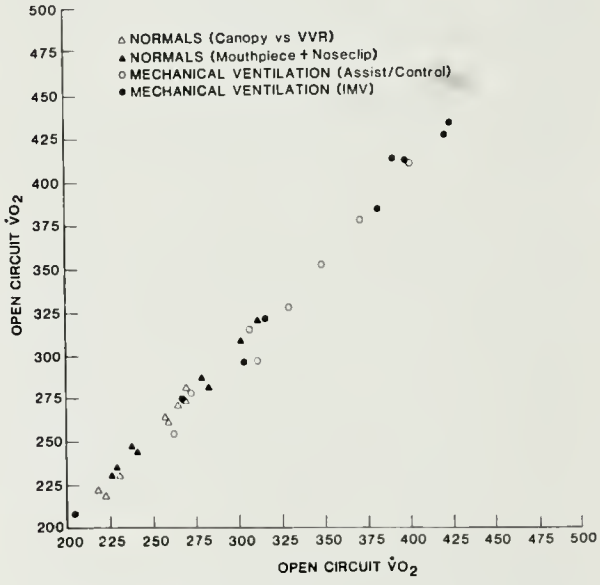

Fig. 12. Correlation of the two $\dot{V}_{O_2}$ measurements made with the Deltatrac. r = 0.994.

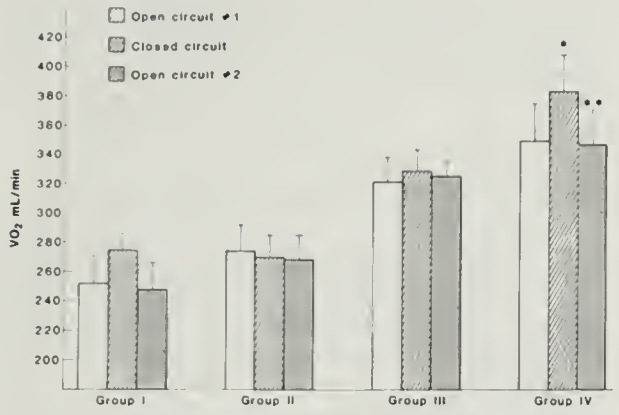

Fig. 13. Comparison of $\dot{V}_{O_2}$ measurements by open-circuit, closed-circuit, and again by open-circuit technique in 4 groups of patients. *$p < 0.05$ OC #1 vs CC, **$p < 0.05$ CC vs OC #2. All other changes not significant. OC = open circuit. CC = closed circuit.

mouthpiece and nose clip on patient comfort and breathing patterns have been previously described.[37] Other investigators studying the effects of breathing via mouthpiece and nose clips versus breathing in a canopy using the same open-circuit system have revealed no differences.[38,39]

Performing Energy Expenditure Measurements

What To Measure?

What is to be measured is resting energy expenditure (REE). As defined by Weissman et al,[40] REE is the energy expenditure that occurs when the patient is lying in bed, awake, and aware of his or her surroundings. Of course, this condition cannot always be achieved with patients in the intensive care unit. The objective is to make the measurement with the patient as quiet as possible so that comparisons with future measurements made under the same conditions will be meaningful. It is important to prevent interaction between patient and caregivers or visitors during the measurement; Weissman et al[40] have shown that significant alterations in $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and REE can occur during routine procedures (Fig. 14). An attempt should be made to perform the measurement during a time when the patient is least disturbed by the environment.

How Long To Measure?

How long the measurement should continue is difficult to pinpoint. If the goal is to measure REE, then the measurement should continue until the

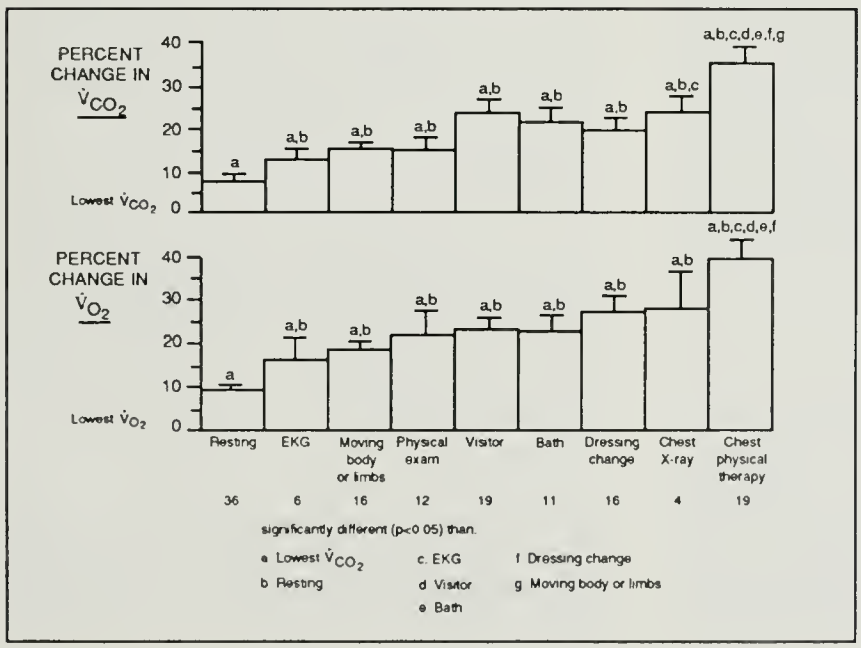

Fig. 14. Effects of routine interactions on $\dot{V}_{O_2}$ and $\dot{V}_{CO_2}$. (Reprinted, with permission, from Reference 40.)

requisite resting state has been maintained for 10 to 15 minutes. This may require that some tests last as long as 2 hours. If, during testing, two periods of rest are observed with similar values for REE, this is ideal. Some indirect calorimeters signal the operator when a 'steady state' has been reached. These signals usually are given when some predetermined criteria have been met. For instance, a calorimeter may define steady state as a change in $\dot{V}_{O_2}$ of $< 5\%$ from one minute to the next. Steady-state markers are useful, but their signaling alone should neither convince the operator that REE has been achieved nor that the test is complete. Only observation of the patient and subsequent comparison of the data can reveal when REE is achieved. The operator must be alert to interruptions that occur during testing. This includes accidental disconnection of the patient from the calorimeter or sudden changes in the patient's status. Some calorimeters utilize artifact suppression to eliminate erroneous data from the measurement caused by leaks or disconnections. This can be a valuable attribute of a calorimeter and may reduce the need for constant observation of the patient during testing.[41]

How Often To Measure?

How frequently to make the measurement is also not completely clear. Weissman et al[42] have studied this and have found that most patients have a day-to-day variation in REE of 5-15%. A smaller group varied REE as much as 50 to 60% from one day to the next. They suggested that the more unstable patients be measured frequently (2 to 3 times/wk) and the stable patients weekly. Unstable patients include those with spiking fevers and hemodynamic instability and those in the immediate postoperative state.

Who To Measure?

The literature is replete with patients at risk for complications from inappropriate or inadequate nutritional support—mechanically ventilated patients,[43] patients with COPD,[44] cancer,[45] renal failure,[46] sepsis,[47] burns,[48] head injuries,[49] and following major surgical procedures.[50] Discussion of each is beyond the scope of the paper and readers are referred to the references for more detail. Lacy et al[51] have developed a list of patients in whom measurements may be valuable (Table 2).

Clinical Application of the Measurement

At present (1990), indirect calorimetry is used to determine caloric requirements of patients requiring nutritional support, to modify nutritional regimens, and to predict successful weaning from mechanical ventilation.

By far, the most frequent application of indirect calorimetry is in the determination of caloric requirements for patients receiving parenteral or enteral nutrition. Numerous published studies have shown the inability of predictive equations to accurately determine caloric requirements of hospitalized patients.[52–57] Each study has shown the nonsystematic fashion in which predictive equations may overestimate and underestimate actual requirements by as much as 50% within the same patient group. The most extensive study of this kind was undertaken by Foster et al,[58] who compared measured energy expenditure to 191 published predictive equations in 100 consecutive hospitalized patients requiring TPN. They found that predictive equations recommended an average of 1076 ± 660 Kcal/day in excess of actual measured REE.[58] Based upon their patient load, they estimated that using predictive equations would have resulted in the administration of 6,947 L of unnecessary TPN/year. By measuring energy expenditure in patients requiring TPN, Foster et al predicted that they would reduce the cost of providing nutritional support by 22%. They concluded that the cost of the indirect calorimeter and personnel to operate it is easily justified based on the substantial cost savings. Measuring energy expenditure to tailor nutritional support regimens is not unlike measuring P_{aO_2} to titrate PEEP or F_{IO_2}. The literature supports the measuring of REE because it is more accurate than predicting REE[52–58] and because it can result in the reduction of nutritional support costs.[58]

Determination of RQ can be used to evaluate substrate utilization and modify nutritional regimens to provide the appropriate balance of glucose, fat, and protein.[59,60] RQ also helps to serve as a quality control. Normally, whole-body RQ resides in a

Table 2. Nutrition Support Department Protocol for Metabolic Cart Measurement*

I. In general, candidates for metabolic cart measurement include:
   A. patients with multiple risk and stress factors which may considerably skew Harris Benedict results.
   B. patients who fail to respond adequately to estimated nutritional needs.

II. More specifically, metabolic cart measurement patient population should include but is not restricted to:
   A. patients in whom multiple nutritional risk and stress factors or disease states affect energy and nutrient requirements or otherwise make Harris-Benedict or other energy equations less accurate. These include patients with:
      1. neurological trauma.
      2. multiple sclerosis, ALS.
      3. amputations, paralysis.
      4. cancer with residual tumor burden.
      5. acute pancreatitis.
      6. COPD.
      7. multiple trauma.
   B. patients in whom height and weight cannot be accurately obtained.
   C. patients who fail to respond sufficiently to estimated nutritional needs.
   D. new, home parenteral nutrition patients.
   E. all intensive care unit patients who remain in critical care units for 5 days duration or longer.
   F. serial measurements for severely hypermetabolic or hypercatabolic patients.
   G. all liver transplant patients and other transplant patients as deemed necessary.
   H. morbidly obese patients.

III. Patients who may be exempt from measurement include:
   A. patients on high frequency ventilation.
   B patients in whom canopy measurements are inappropriate (eg, claustrophobics).
   C. canopy-measured patients who are at significant risk of nosocomial infection (neutropenic; AIDS, reverse isolation).
   D. patients with chest tubes with air leakage.
   E. patients with incompetent tracheal cuffs.
   F. patients in whom $F_{IO_2} < 60\%$ when open-circuit measurement systems are utilized.

*Reprinted, with permission, from Reference 51.

narrow range, 0.67 to 1.3. If results outside this range are encountered, a technical problem exists. Scrutinizing the RQ with respect to the patient's intake can also help alert the operator to possible technical error. The RQ of glucose is 1.0, of fat 0.7, and of protein 0.79-0.82. If a patient not receiving any nutritional support after several days of hospitalization has an RQ of 1.0, a possible technical error should be suspected.

An important but I believe often overdramatized use of RQ is as a guide to modify $\dot{V}_{CO_2}$ through diet. Because the RQ of fat is 0.7, a diet of 50% glucose/50% fat is often recommended for mechanically ventilated patients.[61,62] This allows an adequate number of calories to be delivered at a lower $\dot{V}_{CO_2}$, hence reducing the required $\dot{V}_E$. If

$$P_{aCO_2} = \frac{\dot{V}_{CO_2}}{\dot{V}_A} \times K, \qquad (15)$$

maintenance of normocarbia can be positively or negatively affected by nutritional support. Several authors[63-65] have reported the prolongation of weaning and/or the precipitation of respiratory failure in patients being overfed. This occurs when calories are given in excess of needs, and fat is produced from excess glucose (lipogenesis). When this does occur, RQ will be greater than 1.0-1.3. The excess $\dot{V}_{CO_2}$ requires that the patient increase $\dot{V}_E$; if this is not possible, then the P_{aCO_2} rises. The actual incidence of this problem is probably exaggerated; but, because of its potential impact, it is often implicated when weaning failures are encountered. Monitoring RQ during nutritional support, however, current practice is to deliver a balanced glucose-fat diet to mechanically ventilated patients requiring TPN, unless contraindicated. Pulmocare (Ross Laboratories, Columbus OH) is an enteral feeding developed for patients with COPD that uses a high fat content to help reduce ventilatory work load and still provide a high caloric intake.[66] This product was developed specifically to help deal with the problem of excess $\dot{V}_{CO_2}$. It is important to remember that an $RQ > 1.0$ in patients without respiratory disease is not harmful, although lipogenesis is rarely a goal of nutritional support.

More recently, the use of indirect calorimetry to measure the oxygen cost of breathing (OCB) and predict weaning success has been advocated.[67-69] OCB can be determined by calculating the difference between $\dot{V}_{O_2}$ during spontaneous breathing and $\dot{V}_{O_2}$ during controlled ventilation.[70] McDonald et al[67] found that OCB correlates with the duration of weaning.

The greater the OCB, the longer the patient may require mechanical ventilation. Lewis and colleagues[68,69] have suggested that OCB may be a more sensitive predictor of weaning success than are conventional measurements. They found that patients who were weaned successfully had a change in $\dot{V}_{O_2}$ between spontaneous ventilation and controlled ventilation of 7.7 ± 8.8% and those who failed to wean of 24.7 ± 12.3%.[69] Kemper et al[71] have also studied this application of indirect calorimetry, but have found no correlation between changes in $\dot{V}_{O_2}$ or $\dot{V}_{CO_2}$ and weaning success.

Some of the current literature suggests that determining nutritional requirements and monitoring patient response to therapy with indirect calorimetry is warranted. Modification of nutritional regimens by determination of RQ is also helpful in patients with respiratory disease. However, the use of $\dot{V}_{O_2}$ measurement to determine weaning success needs further study.

A Final Note

Indirect calorimetry is often criticized because of the difficulties encountered in its use and because of the apparent lack of effect.[72] It has been my experience that nutritional support requires the often-discussed, little-practiced team approach—a physician interested in the measurement must be backed up by nutritionists and dietitians capable of implementing nutrition, a respiratory care practitioner capable of performing indirect calorimetry in the presence of an ever-increasing array of sophisticated equipment, and a nursing staff in tune with the nutritional needs of their patients. It is unreasonable to expect nutritionists to learn the intricacies of mechanical ventilators and their various modes. To do so is to invite failure. On the other hand, a respiratory care practitioner with a functioning calorimeter but no support from the nutritionists in interpretation and application of the data will meet the same fate.

Lastly, the evidence for successful nutritional support is not measured in hours or days but weeks. There is no instant gratification gained from initiating nutritional intervention, and this lack of immediate feedback appears to hinder its use. I encourage respiratory care practitioners to become involved in this aspect of patient care that requires technical skill, knowledge of nutrition-patient interactions, and patience. I believe that both patient and therapist will benefit.

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Branson Discussion

Rodriguez: You indicated that to use indirect calorimetry with mechanical ventilation the circuitry might have to be different on different ventilators. Are you aware of any schematics available—from manufacturers of ventilators or calorimeters—to assist the respiratory care practitioner in assembling this special circuitry?

Branson: I think the thing to do is to look at the manuals for each ventilator. You need to look at two things—the first is the blender mechanism. Systems such as the Hamilton and Advent systems have regulators for blenders rather than proportional valves and a reservoir that serves as a mixing chamber. The chamber mixes the gases so that when the gas exits the ventilator the F_{IO_2} is very stable, unlike the gas exiting ventilators that employ proportional valves (such as the Puritan-Bennett 7200). The second thing is the expired side. The Bear 5 uses a continuous bias flow to zero the flow transducer. There are no vortices at zero flow; so 8 L/min runs through the system constantly and the flow transducer 'thinks' that zero. This bias flow improves the accuracy of the volume measurement at low flows. But, that 8 L/min (collected from expiration valve) is going to end up in the calorimeter and is going to dilute F_{EO_2} and F_{ECO_2} values and increase minute ventilation. These are things that you have to be aware of when you are doing calorimetry on mechanically ventilated patients. You have to know how the ventilators work with respect to controlling F_{IO_2} and the presence of any continuous flow.

Rodriguez: Are you aware of any special precautions that one should take in determining the caloric requirements of burn patients on mechanical ventilation?

Branson: With respect to what?

Rodriguez: With respect to any special metabolic problems that might be caused by topical antibiotics, for example.

Branson: Not to my knowledge.

Chatburn: Can any of the devices that you have described be used with neonatal ventilators? Are you aware of any work that has been done with neonates?

Branson: None of the devices that I described can be used with neonates. I know there are a lot of companies working toward this end, but the basic problem lies in the fact that neonates use uncuffed tubes, and if you can't collect all of the expired CO_2 then you can't perform indirect calorimetry. I don't know if it will ever be conquered technically because it is a physiologic problem.

Marini: I like the idea of paying attention to nutrition, but I have trouble understanding the value of missing my estimate by as much as 33 or 40 or 50% on a given patient. If you do some calculation about how much weight a person would lose over an extended period of time, it seems trivial. If you provide an adequate number of calories to a patient (something over 1200 calories/day) and an appropriate nutrient mix, I would anticipate that the effects on ventilatory drive, muscle strength, muscle function—weaning status—that we might otherwise worry about in a starving patient are more or less put on hold. Maybe it's just my ignorance of the topic, but it seems to me that indirect calorimetry measurements are highly complex. They require an exact and precise evaluation by skilled professionals to really give you numbers that mean anything or come close to what the actual values are. Why not just give people about 1800 calories/day and see what happens? I know that this seems anti-scientific, but from a practical point of view I think it makes a lot of sense.

Branson: From a practical point of view, I can see doing that except in the cases in which we need to provide all of the patient's nutritional support for more than 4 or 5 days. One of the things about nutrition is that it can never be made up—once you are 2000 calories behind, you are 2000 calories behind! You can't make it up by giving an extra 2000 calories the next day; there is a limit to how much extra you can give. Some of the work that has been done at the University of Michigan by Bartlett[1] suggests that caloric deficit (based on the patient's measured energy expenditure vs what the patient actually gets, which is another important thing to look at) correlates with mortality. Don't look at what is ordered, look at what the patient actually receives. Those things are related to mortality. We have found that adequate nutritional support, along with electrolyte supplementation (eg, magnesium and phosphorus), helps to wean long-term ventilator patients—who have experienced muscle wasting—off of the ventilator. I think if you empirically state: "We'll give this much," but the patient's actual needs are greater, then after a period of a week you are going to be way behind. And, it will be very difficult to make it up. I don't know that watching body weight actually gives you a true picture of what's happening, because you don't know how much weight change is related to nothing at all except fluid shift. I understand what you're saying, but I think that indirect calorimetry is a much more accurate technique. You would not provide oxygen to a patient without drawing an arterial blood sample! There need to be some good studies that look at 'early' nutrition in mechanically ventilated patients. These type of studies have been done with surgical patients, and it has been shown that preoperative nutrition or perioperative nutrition have decreased mortality in certain patient groups.[2,3]

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Marini: I submit that there are many fully active, healthy people in this country who go on intermittent 1000-1200 calorie diets—in fact, they might even improve their health as long as they're getting a balanced diet and a modest number of calories. I think that we have a lot more reserves, in terms of compensation for inadequate caloric intake, than some people suggest. That's just my viewpoint!

Branson: I think it depends on the patient! I find that chronic lung disease patients don't have much reserve at all. We know that in the COPD population decreasing body weight (or percent of body weight) is associated with increasing mortality.[1] Most of the patients we see come in malnourished—either because of low socioeconomic status or chronic illness. We don't start out with someone who is healthy! I think in healthy people we can make the assumptions that you alluded to, but I'm not so sure that we can in debilitated patients.

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Krieger: To follow up on John's (Marini) question—Do you think that giving calories and then looking at nitrogen balance is a reasonable compromise?

Branson: No—I think that reliable nitrogen balance measurements are difficult to make. If you try to predict 24-hour nitrogen based on a 2-hour nitrogen measurement, you may be off as much as 30 or 40%.[1] I think a lot depends on the patient. Our experience has been that nitrogen balance is a

slow-to-resolve, slow-to-catch-up type of measurement; and it doesn't give you the type of information that you need.

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East: If a small, accurate, inexpensive noninvasive calorimeter were available—one that you could afford to put at each bedside (perhaps costing $2,000)—do you think it would be useful to you in terms of helping you adjust the ventilator or make decisions during weaning?

Branson: I didn't consider this in my presentation, because obviously there was too much to cover already. But, at the AARC meeting we did talk about using the true oxygen cost of breathing as a potential weaning technique. The true oxygen cost of breathing is the oxygen cost measured during total spontaneous breathing minus that measured during total controlled ventilation. I think a lot of people have bastardized it to be the difference between the $\dot{V}O_2$ during pressure support and the $\dot{V}O_2$ during assist control or the $\dot{V}O_2$ during CPAP and have compared the differences. The study by McDonald et al[1] suggests that measuring the oxygen cost of breathing is useful in weaning patients, but the study by Kemper et al[2] suggests that oxygen cost of breathing doesn't affect patient weaning success—so I'm not sure! If you have continuous DO_2 measurement and some type of invasive S_{aO_2} or $S_{\bar{v}O_2}$ measurement, you might be able to get online cardiac output, which might prove useful in the management of some patients (eg, CICU patients after coronary artery surgery).

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Hess: Rich, what about the 150- or 200-bed hospital that can't afford to buy one of these $30,000 instruments, but wants to take a Douglas bag, a spirometer, and a gas analyzer and do their measurements that way?

Branson: We performed Douglas bag collections before we had our first calorimeter. But, if you think indirect calorimetry is hard with the machine that comes from the manufacturer, you will be amazed at how hard the Douglas bag technique is! Dean (Hess), I know you have used the Douglas bag technique. I think it's a viable alternative; if I couldn't afford a calorimeter, that's what I would do.

Hess: I've done it both ways, and it's not that easy either way, actually!

Burke: Just a point on getting this technology to move forward—we know there's a wealth of data that says that with animals (Zucker rats) there is a wide variety in the efficiency in laying down fat.[1,2] We use equations in which we say if respiratory quotient (RQ) is 1.0 you're breaking down carbohydrate, and if it's 0.7 it's fat. Well it's probably not that easy! Even among us in this room, we have different efficiencies for laying down fat, which just points out that this really needs to be looked at by professionals who know what they're doing, know what they're measuring, know the patients, and who will discuss the results with the physicians so that meaningful changes in therapy can be made. There's still a lot to learn here; we really don't know what goes on in humans.

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2. Landau BR, Wahren J. Quantification of the pathways followed in hepatic glycogen formation from glucose. FASEB J 1988;2:2368-2375.

Branson: I agree, but I think we have to start somewhere. And respiratory quotient is a good quality control because regardless of how efficient or inefficient a person is, an RQ below about 0.67 or above 1.3 cannot be achieved (though a rat can have an RQ of 9.0).[1] So, I think in humans we do know what a physiologic range is, which is very helpful.

1. Cherniack NS, Longobardo GS. Oxygen and carbon dioxide gas stores of the body. Physiol Review 1970;50:196-204.

Kacmarek: I tend to agree with what John (Marini) said concerning the problems associated with performance of the study. We are doing indirect calorimetry, and what concerns me is how accurate our results are and the cost benefit for all the effort and time that I'm putting into having the practitioners perform the study? As you've already indicated, I have to take one person and have them do the indirect calorimetry. If they can get 4 to 6 studies done a day they're doing very well! What kinds of patients really need calorimetry? According to the data that you presented and to what has been said thus far in the discussion, I would assume that you are of the opinion that most surgical patients who are mechanically ventilated need indirect calorimetry. I just can't believe that's true! Based upon my experience, I would think that only a limited number of patients really benefit from this noninvasive monitoring and the consequent modification of their feeding regimens. On which patients should we perform indirect calorimetry?

Branson: We select them based on whether they're getting some type of artificial nutrition. Mechanically venti-

lated patients who are going to receive some type of artificial nutrition for an extended period of time (eg, patients who have major gut resections or patients following trauma who you know for physiologic reasons will not be able to be fed enterally or to feed themselves for an extended period of time) need to have this type of measurement done. Again, you're not going to deliver oxygen without measuring oxygen status, so why should you provide nutritional support without measuring nutritional requirements? We can argue about the leeway that we have with nutrition, but I don't think we know if we have any leeway with nutrition—we just assume it! At my institution, we perform calorimetry on: (1) patients in the ICU who are going to be mechanically ventilated for a week or more and (2) on patients on the floors who have undergone major surgery and are going to be sent home on total parenteral nutrition (TPN). Patients who will be depending on you to give them all of their nutrition need to be measured—you need to make an accurate estimate of their nutritional needs! I think that you can modify some nutritional regimens based on the respiratory quotient to decrease CO_2 production (by supplying a greater portion of calories with fat rather than glucose). You can anticipate that CO_2 production is going to increase as you increase the number of calories given, and you can assist the patient by providing a greater amount of mechanical ventilation.

Kacmarek: So, you're using 7 days essentially as the cutoff for noninvasive calorimetry in mechanically ventilated patients?

Branson: Yes! Otherwise the patient will be off the ventilator in a few days, eating on his own, and won't need that type of measurement!

Measuring and Monitoring Lung Volumes Outside the Pulmonary Function Laboratory

David J Pierson MD

Lung Volumes: Measuring vs Monitoring

Total lung capacity (TLC) and its subdivisions, as shown schematically in Figure 1,[1] are affected in well-characterized ways by different disease processes. Obstructive lung disorders such as asthma and emphysema tend to hyperinflate the lungs in proportion to the severity of the obstruction, as reflected by increases in TLC, functional residual capacity (FRC—lung volume at the end of a normal tidal exhalation), and residual volume (RV—the volume of air remaining in the lungs after a maximal exhalation). Conversely, restrictive disorders, such as pulmonary fibrosis and respiratory muscle weakness, are manifested by decreases in TLC, FRC, and RV.

Measuring lung volumes is a routine part of the evaluation of clinically stable patients in the pulmonary function laboratory[2], but such measuring is not routine for the evaluation of individuals who cannot conveniently or safely go to the laboratory (eg, those in the intensive care unit [ICU], emergency ward, or operating room). This article summarizes the desirability, availability, and practical limitations of lung-volume measuring techniques that can be performed in nonlaboratory sites, focusing mainly on the ICU and on mechanically ventilated patients.

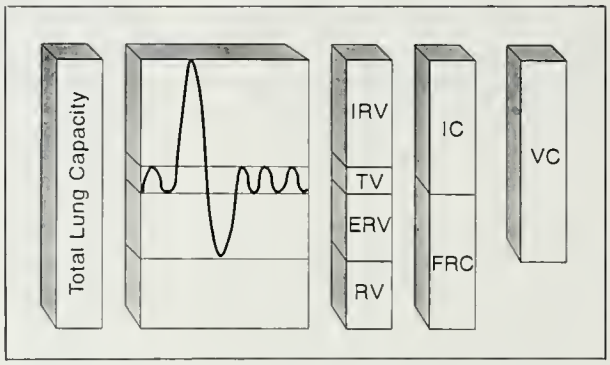

Fig. 1. Total lung capacity (TLC) and its subdivisions. Individual volumes (inspiratory reserve volume [IRV], tidal volume [V_T], expiratory reserve volume [ERV], and residual volume [RV]) combine to make up capacities (inspiratory capacity [IC], functional residual capacity [FRC], vital capacity [VC], and total lung capacity [TLC]). (Adapted, with permission, from Reference 1.)

In a discussion of the measuring of lung volumes in the context of a conference on noninvasive monitoring, it is important to distinguish at the outset between 'measuring' and 'monitoring.' They are not the same—a technique well suited to the former may be entirely inapplicable to the latter. At a previous Respiratory Care Journal conference that focused upon the monitoring of critically ill patients,[3] considerable effort was devoted to developing a definition of monitoring in this context. A comprehensive though admittedly awkward definition was offered by Hudson[4] in his conference summary:

> Monitoring is making repeated or continuous observations or measurements of the patient, his or her physiological function, and the function of life support equipment, for the purpose of guiding management decisions, including when to make therapeutic interventions, and assessment of those interventions.

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At the same conference, George[4] described monitoring more concisely as "the gathering and analysis of data on an ongoing basis for the purpose of guiding management decisions." Three distinguishing characteristics of monitoring are thus the measurements themselves, the repetitive nature of the measuring, and the guiding role that the measurements play in the ongoing evaluation and management of the critically ill patient.

In this review, as I describe the techniques by which lung volumes may be measured outside the pulmonary function laboratory, I will also comment on the feasibility and practical value of such procedures in the context of these definitions of monitoring.

Which Lung Volumes Are Desirable To Monitor and Why?

Most respiratory disorders encountered in the ICU markedly affect lung volume. The adult respiratory distress syndrome (ARDS), cardiogenic pulmonary edema, recent upper abdominal surgery, and acute lobar atelectasis result in acute restriction of lung volume. Status asthmaticus and severe, acute exacerbations of chronic obstructive pulmonary disease (COPD) worsen the underlying hyperinflation characteristically present in patients with these conditions. Air trapping during mechanical ventilation (caused by incomplete exhalation between positive-pressure breaths and manifested by endogenous, or intrinsic, positive end-expiratory pressure [auto-PEEP]),[5] is a common and serious complication not confined to patients with airway obstruction.

Because the basic pathophysiologic mechanisms in these processes are tied directly to changes in lung volume, the ability to monitor such changes quantitatively is desirable. To use ARDS as an example, the therapeutic use of positive end-expiratory pressure (PEEP) is tied to its presumed effects on lung volume, especially FRC.[6-8] The variables commonly monitored during PEEP therapy (eg, lung-thorax compliance, arterial oxygen tension [P_{aO_2}], and cardiac output) vary, presumably because of the effect that PEEP has upon lung volume. To monitor FRC directly rather than indirectly is desirable both conceptually and practically because it may eliminate costly or less pertinent measurements.

Abbreviations Used in this Paper

ARDS	— Adult respiratory distress syndrome
auto-PEEP	— Endogenous or intrinsic PEEP
COPD	— Chronic obstructive pulmonary disease
CPAP	— Continuous positive airway pressure
F_{IO_2}	— Fractional inspired oxygen concentration
FRC	— Functional residual capacity
ICU	— Intensive care unit
IMV	— Intermittent mandatory ventilation
P_{aCO_2}	— Arterial carbon dioxide tension (pressure)
P_{aO_2}	— Arterial oxygen tension
PEEP	— Positive end-expiratory pressure
RIP	— Respiratory inductive plethysmography
RV	— Residual volume
SF_6	— Sulfur hexafluoride
TLC	— Total lung capacity

A Guide to the Use of SI in this Paper*

The SI unit for pressure is the kilopascal (kPa).
(cm H_2O)(0.098 06) = kPa.

*For further information on SI (le Systeme International d'Unites), see Respir Care 1988;33:861-873 (October 1988) and Respir Care 1989;34:145 (February 1989—correction).

It might at first seem that TLC should be as important an assessment tool as FRC, but it is not. To determine FRC, it is necessary only to allow the ventilatory apparatus to come to rest with an open airway. To measure TLC during mechanical ventilation, the lung must be distended to some arbitrary level because the patient's own maximal effort may not result in the greatest possible lung volume. Mechanical inflation of the lungs can be carried out to a preselected peak inflation pressure, but volume varies as lung-thorax compliance is altered by disease or therapy.

The potential drawbacks associated with the measuring of TLC in mechanically ventilated patients are also associated with the measuring of RV in this population—the measuring of either of these variables requires mechanical manipulation of the patient's ventilatory apparatus and thus reflects the actions of the clinician as much as the state of the patient. For

these reasons, FRC is the lung volume best suited for measuring and monitoring in the ICU. In order to be suited for monitoring, the FRC measuring technique must be rapid, simple to carry out, and clinically accurate.

Measuring FRC in the Intensive Care Unit

The potential value of being able to accurately measure FRC in the ICU (especially during mechanical ventilation) is evidenced by the number of different procedures and techniques that have been designed for this purpose and described in the literature.[9-36] A number of investigators have reported results that are clinically acceptable, although perhaps not as precise as those obtainable under the controlled conditions of a pulmonary function laboratory.[2]

Closed-Circuit Techniques

With the closed-circuit method, an indicator gas is introduced into the system consisting of the ventilator circuit and the patient's lungs and becomes distributed throughout this system during a period of rebreathing. If the original concentration and volume of the indicator gas added to the system are known and the new equilibrated indicator concentration is measured, then the total volume in the patient's lungs and ventilator circuit can easily be calculated. Subtracting the volume of the ventilator circuit from the total system volume gives the volume of gas that was present in the patient's lungs at the moment the rebreathing started. Because FRC is believed to be the most 'natural' and reproducible lung volume, closed-circuit techniques (and the other techniques to be discussed) attempt to measure FRC rather than TLC.

The indicator gas most commonly used is helium because it is readily available, harmless in the low concentrations used, and easily measured. Because an increase in arterial carbon dioxide tension (P_{aCO_2}) during rebreathing would stimulate ventilation and could possibly cause patient discomfort and intolerance, carbon dioxide is removed from the system by absorption into soda lime. To prevent a continuous loss of volume from the system, oxygen is generally bled in at a rate intended to compensate for the patient's oxygen consumption.

The techniques used for closed-circuit helium dilution in the ICU[9-15] are the same in concept as those used in the laboratory. Suter and Schlobohm[12] in 1974 reported a 'bag-in-a-box' technique that was conceptually simple, although it cannot readily be duplicated from the information provided. In 1981, my co-workers and I[15] reported essentially the same technique, but described the apparatus in more detail and used components that were readily available from commercial sources and easily accessible to the staff of a general hospital (rather than being specially fabricated or modified equipment designed for research). That setup is depicted in Figure 2. It enables FRC to be determined on whatever ventilator settings are being utilized, in either the assist-control or intermittent-mandatory-ventilation (IMV) modes, and may be used with or without PEEP. A minimum of 30 minutes is required to measure FRC with this technique.

Open-Circuit Techniques

Using the open-circuit approach, a known amount of indicator gas is added to the patient's inspired gas, its concentration in the expiratory circuit is monitored breath-by-breath until it is constant, and the volume required for this equilibration to take place is determined. The volume of distribution (ie, FRC) can then be calculated. In these techniques, the indicator gas may be helium, oxygen, nitrogen, or sulfur hexafluoride. A main feature of these open-circuit techniques is their relative simplicity in comparison to the closed-circuit methods.

Hylkema and colleagues[16] used a helium-dilution technique that alledgedly required no bulky equipment and did not disturb the patient at the endotracheal tube connection site. They administered a gas mixture consisting of 50% helium and 50% oxygen until equilibrium was reached (taking about 2 minutes in each of their 10 patients), and then switched to 100% oxygen until washout was complete (another 2 minutes). The results agreed within 10% with the results obtained with the 'bag-in-a-box' system of Heldt and Peters,[14] but the method could not be used in patients requiring supplemental oxygen of high concentration and, as the authors pointed out,[16] the method would not be accurate in patients with poorly ventilated lung regions.

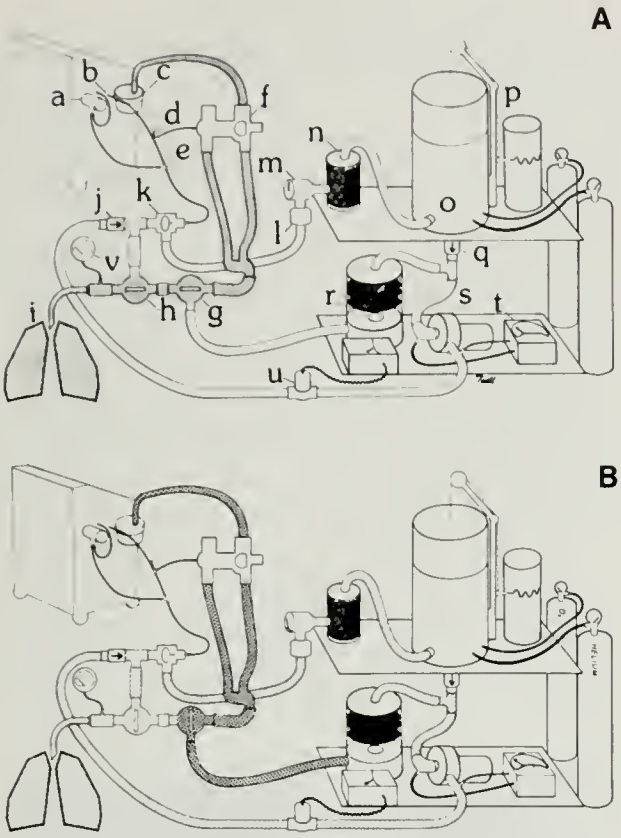

Fig. 2. A setup for closed-circuit helium-dilution determination of FRC in mechanically ventilated patients. A, apparatus prior to switching the patient to the 'bag-in-a-box' circuit: a—ventilator and PEEP device; b—spirometer dump nipple; c—ventilator humidifier; d—three-way stopcock; e—T-connector; f—ventilator circuit manifold; g & h—Collins three-way respiratory valves; i—patient; j—Bird one-way valve; k—exhalation valve; l—bacteria filter; m—Wright spirometer; n—CO_2 absorber; o—inlet port of Collins water-seal spirometer; p—Collins residual volume apparatus; q—Bird one-way valve; r—Bird Mark VI 'bag-in-a-box;' s—motor blower; t —helium meter; u—O_2 analyzer; v—pressure manometer. The shaded path represents the initial gas circuit with the patient being mechanically prior to switching to the bag-in-a-box circuit for FRC measurement. B, path of gas flow during FRC measurement: dark stipples—ventilator to bag-in-a-box circuit; light stipples—patient helium-rebreathing circuit. (Reproduced, with permission, from Reference 15.)

A nitrogen-washout technique, utilizing two electronically-synchronized ventilators and an online computer, was described by Paloski et al.[17] Their apparatus is diagrammed in Figure 3. Tested on a mechanical lung simulator and also on 18 ventilated patients, the method produced a good correlation between paired sets of measurements. Further, the authors stated that ". . . the system is easy to set up and operate . . . [and], therefore, provides safe, convenient, and accurate measurements and is routinely used in critically ill patients receiving ventilatory support in either control, IMV, or CPAP modes."[17] Whether the last statement remains true at the authors' institution or elsewhere is unknown.

Mitchell and associates[18] reported a technique that employed oxygen washin rather than direct nitrogen washout. They made a 20% or greater step-change in inspired oxygen fraction (F_{IO_2}), and used a computer-based respiratory monitoring system with a fast-response oxygen analyzer and respiratory flowmeter. This technique is an indirect version of the nitrogen-washout method. Results were reproducible and correlated well with FRC determined by helium dilution. As with several of the other methods reviewed here, the authors consider their

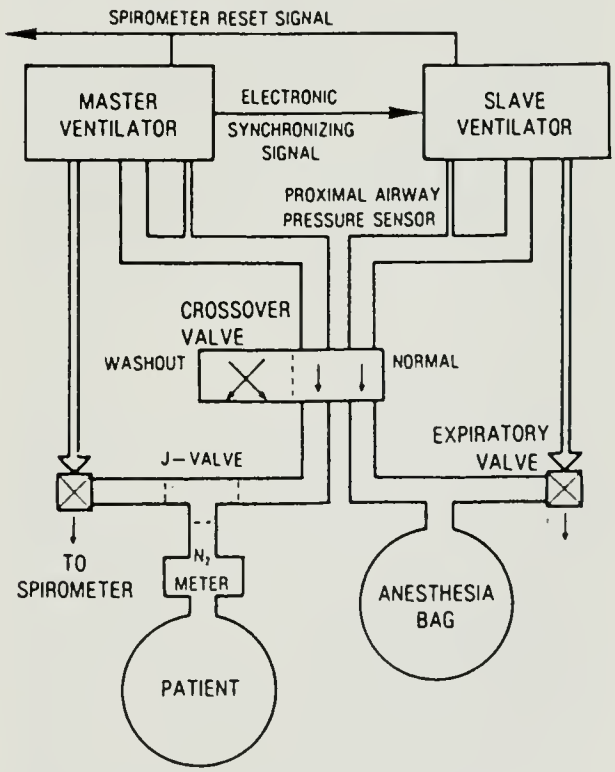

Fig. 3. Apparatus for open-circuit nitrogen-washout determination of FRC during mechanical ventilation. It utilizes a second ventilator, electronically synchronized with the patient's ventilator, and requires an online computer. (Reproduced, with permission, from Reference 17.)

technique to be both accurate and practical for routine clinical application: "With this approach, FRC may be measured on a routine basis in the ICU without disconnecting the patient from the ventilator . . . and has the potential for wide application."[18]

One potential difficulty with clinical implementation of methods relying on a step-change in F_{IO_2} is the relatively large 'washout volume' of many ventilators. Benson and I[19] studied four different ICU ventilators and found that, depending on the tidal volume and cycling frequency used, the intended new F_{IO_2} might not actually be delivered to the patient for as long as 2 minutes after the ventilator controls are changed. A subsequent study using later-generation ICU ventilators, reported in an abstract from our institution, had similar results.[20]

A Scandinavian group[21,22] has described a method of determining FRC during mechanical ventilation in which the inert gas sulfur hexafluoride (SF_6) is administered at low concentration by an open-circuit technique. The amount of SF_6 present in the patient's lungs at the end of a washin period is calculated during washout (from tracer gas concentration and expired flow); lung volume is derived mathematically. These investigators report good agreement between FRC determined by their method and FRC determined by nitrogen washout in mechanically ventilated patients; whether this technique has found clinical application in patient management is not stated.

Radiographic Techniques

A completely different approach, in which lung volume is derived from the chest roentgenogram (using assumptions about thoracic geometry), has at least theoretical application in the ICU setting.[2] If the height, width, and depth of the lung can be measured, then theoretically the calculation of its volume should be possible. Hurtado and Fray[23] first attempted to do this a half-century ago, and in 1960 Barnhard and associates[24] reported a refinement of the method that required complicated calculations but was more applicable than the original. In the years since that time, numerous investigators have described refinements and variations of this technique.[25–32]

In the late 1960s, a planimetric method for determining lung volume from chest roentgenograms was introduced by Pratt and associates.[25,26] This method employs a device called a compensating polar planimeter that traces the boundaries of the lungs on anteroposterior and lateral radiographs and measures the surface area that they cover. A regression equation is then used to calculate lung volume from the sum of the areas in the two projections. In a study published in this journal, Wehr and Masferrer[27,28] evaluated the clinical applicability of this technique in 11 patients with obstructive and restrictive lung disease, comparing planimetrically-derived TLC to that measured in a body plethysmograph. They found good agreement between results of the two techniques, and pointed out that FRC and other subdivisions of TLC can be determined easily if a simultaneous spirogram is obtained.

Application of the planimeter to the frontal roentgenograph is shown in Figure 4.[27] More recently, computer assistance[31,33] and the use of calculations derived from computed tomographic cuts of the lung[34]

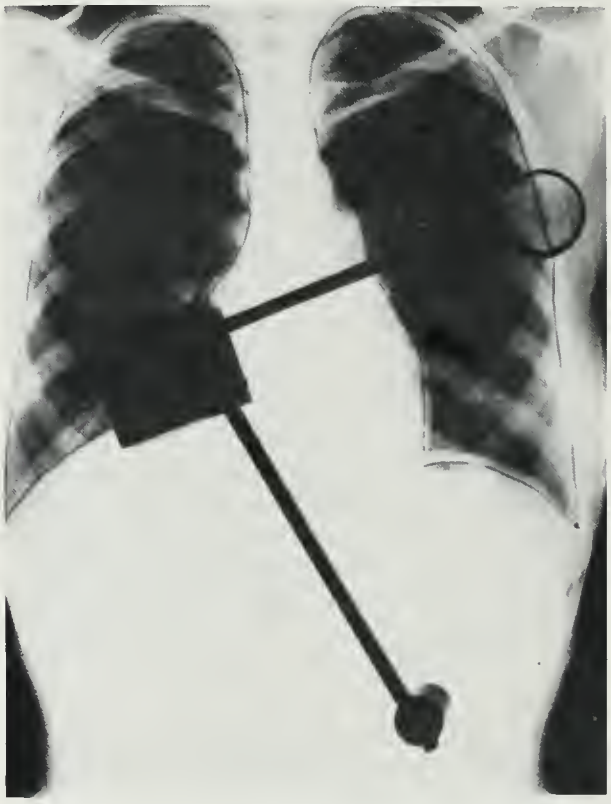

Fig. 4. Frontal chest roentgenogram overlaid by a compensating polar planimeter for radiographic determination of lung volume. (Reproduced, with permission, from Reference 27.)

have been added to further refine the radiographic approach to measuring lung volume, but it is uncertain whether this has increased the bedside applicability of the method.

The radiographic techniques described so far have employed technically acceptable, 6-ft roentgenograms performed on normal subjects and on clinically stable patients. Some investigators have attempted to apply the radiographic approach outside the laboratory (in a manner more closely approaching monitoring).[32,35-37] Rothstein and associates[37] successfully measured TLC radiographically in 32 patients experiencing acute asthma attacks, although these subjects were not in the ICU and were able to perform breath-hold maneuvers while standard chest radiography was performed.

Ries et al[36] demonstrated that reasonably accurate determinations of planimetric lung volume in normal subjects could be made in the supine position, using posteroanterior and lateral portable roentgenograms of the chest. Because of the theoretical desirability of measuring FRC during sleep (eg, while studying the physiologic effects of sleep apnea), Block and colleagues determined FRC by the planimetric method using frontal and lateral roentgenograms of the chest simultaneously obtained during sleep.[35] Their results are considered clinically meaningful, but the requirement for lateral films and the logistic difficulties of obtaining repeated, reproducible films in critically ill patients make successful transfer of this technique to the ICU a doubtful prospect.

Several factors make the radiographic approach poorly suited for the monitoring of lung volumes in the ICU, especially in mechanically ventilated patients. Foremost among these are the practical difficulties of obtaining reproducibility—in FRC, patient positioning, and bedside radiographic technique—during the assessment and management of the critically ill patient. The artificiality of TLC determined during mechanical ventilation has already been alluded to; however, FRC may also vary breath to breath and may be difficult to capture in an agitated or tachypneic patient. Because the radiographic method is based on chest geometry, precise anteroposterior alignment of the patient is crucial to its accuracy; avoidance of motion artifact is also crucial. The difficulty in maintaining correct patient alignment and avoiding motion artifact increases as the stability of the patient decreases.

Attempts to abbreviate the cumbersome planimetric procedure established by Pratt and associates,[25,26] by using a single set of three measurements made on a specially-exposed X-ray film, resulted in a marked loss of precision. This prompted the investigators to conclude that "the rapid radiographic method of estimating total lung capacity can make little contribution to the diagnostic assessment of lung disease."[32] In addition, radiography performed amidst the hubbub at the bedside of an unstable critically ill patient—which unfortunately is the very setting in which lung-volume monitoring is likely to be most valuable—is far less precise than radiography performed in the more controlled environment of a radiology department.

Monitoring Changes in Functional Residual Capacity

The monitoring of tidal volume and vital capacity (or at least inspiratory capacity) in intensive respiratory care is important, but it is covered elsewhere in this conference,[38] in the proceedings of the 1984 RESPIRATORY CARE Journal conference on ICU monitoring,[3] and in the proceedings of last year's Journal conference on pulmonary function testing.[39] Although the measuring of absolute lung volume is the primary focus of this article, the monitoring of changes in FRC during the course of an illness or in response to interventions[6,7] could certainly be valuable—even if the actual FRC itself cannot be quantitated.

Respiratory inductive plethysmography (RIP) was used in a recent study[40] in an attempt to find an alternative method of detecting auto-PEEP; during this study, changes in FRC were monitored. As shown in Figure 5, when external PEEP was increased incrementally from 0-4 cm H_2O [0-0.4 kPa] (at "1") to 4-6 cm H_2O [0.4-0.6 kPa] (at "2"), no change representing thoracic and abdominal motion occurred in the monitor tracings. This indicates that FRC remained constant and that the patient's auto-PEEP exceeded the amounts of externally applied PEEP. However, when PEEP was increased to the level of 6-8 cm H_2O [0.6-0.8 kPa] (at "3"), the tracings were deflected upward, indicating an increase in end-expiratory lung volume (FRC) and signifying that the patient's auto-PEEP was just less than the externally-applied PEEP. Although it was not

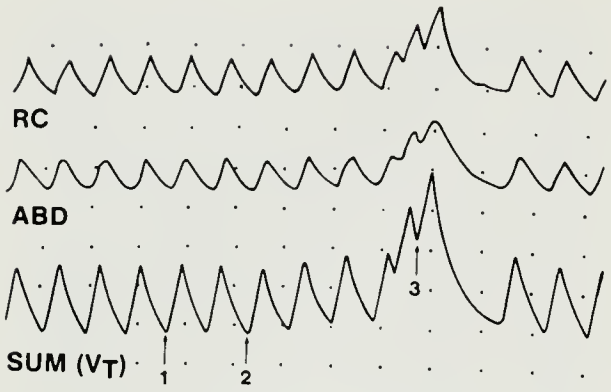

Fig. 5. Tracings obtained by respiratory inductive plethysmography (RIP), showing the effect of incremental additions of external PEEP (at Points 1, 2, and 3) on FRC, as part of a technique for detecting and quantitating endogenous PEEP (auto-PEEP). (For further explanation see text.) (Reproduced, with permission, from Reference 39.)

reported directly, with proper calibration of the device this deflection can be quantitated as the amount of change in FRC.

RIP has also been shown to track tidal volume in spontaneously breathing patients.[41] Although it remains an indirect, semiquantitative indicator of FRC, RIP has potential for wider application as an ICU monitoring technique,[42] particularly in view of recent simplifications in the calibration procedure.[43]

Conclusions: Good News and Bad News

Numerous investigators have demonstrated that FRC can indeed be measured in the ICU in mechanically ventilated patients (at least with accuracy acceptable for patient management). However, the very number of individual systems, techniques, and 'improvements' on previous methods reported by these investigators suggests that a problem still exists. The measuring of lung volumes outside the pulmonary function laboratory is still too complicated, too cumbersome, and too time- and labor-intensive to be practical for routine application. Although several authors have pointed out how rapidly their methods permit the measurements to be made, in practice the procedures are still too involved to be performed repeatedly over a period of a few hours. It is my perception that these techniques are rarely used for patient management, even at the centers where they were developed and reported.

This could change as ventilator and monitor manufacturers begin to build these methodologies into their products and as clinical application becomes easier and more automated. In theory, indicator-gas washout capabilities could be incorporated into devices already at the bedside, and, if acceptably reliable (and not prohibitively expensive), the measuring of FRC could move closer to a true monitoring technique. However, this is still pure speculation and if any of the techniques described in this article do become more conveniently available, they will need to be subjected to clinically relevant, prospective examination to determine whether they do in fact augment the clinician's monitoring armamentarium.

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Pierson Discussion

East: Our group designed and constructed the SF_6 FRC-measuring system that Siemens will be bringing out as a product shortly. It's been evaluated in COPD and ARDS patients, and it does work very nicely. It's fully automated so it is easy to use, and you can get measurements at specific intervals (like 15-min intervals) automatically. The cost is about 25 cents per measurement (including the cost of the equipment). It doesn't add a lot of dead space or hardware to your system, and it will work with any mode of mechanical ventilation. The only bad thing we've seen in terms of

its applicability in ARDS patients is that if you have any gas leaks, all bets are off—so if you've got chest tubes or other kinds of problems, the gas dilution techniques don't work very well. And the density of sulfur-hexafluoride gas (SF_6) is a bit of a problem if you have equilibration problems. So, SF_6 may require prolonged equilibration time (compared to helium in COPD patients, for instance) to get reliable measurements.

Pierson: In your experience with that technique, how long does it take to actually make the measurement, and how much labor is involved on the part of the bedside person?

East: It will run independently of anyone being there. The measurement is fully automated. You can schedule it at 15-min intervals; and it will do it, record it, and store it out on the PC disc—you don't even have to be there!

Pierson: Will it tell you whether the patient is lying on his side or whether he's just coughed or been suctioned, or . . . ?

East: If you want to do it by hand, you can do it by hand. We were curious about what would happen if you just set it up and let it run. You can do it on a manual activation basis too; and there's a place to put information in about the status of the patient, but we were just curious about what you would get if you used it at routine intervals. What you get if you move people around and do all kinds of maneuvers is a lot of noise—as you'd expect! So, your final conclusion may be true—in ARDS patients it may be hard to make a lot of sense out of the FRC measurements. But, it is a nice system—it certainly makes it easy to do!

Pierson: And it won't make the ventilator any more expensive, will it? [laughter from the group]

East: I have no idea about that.

Mathews: With respect to the planimetric techniques, a few years ago, in one of our research-methods courses, we developed a technique that, although marginal at that time, might have some application now. We used an Apple II computer with a graphics tablet to trace x-rays, and we were primarily looking at pneumothoraces. We were able to integrate and differentiate the curves to come up with some numbers that were pretty good. The technique as David (Pierson) pointed out earlier is heavily dependent upon radiologic technique rather than the computational skills or the computer expertise of the researcher. However, I would think that by consistently using the same radiographer that this technique might prove to be valuable.

Pierson: I think that, as with calorimetry in the critically ill patient, the measurement of lung volume is especially susceptible to the 'garbage in—garbage out' problem.

Mathews: I concur!

Hess: One of the points that sort of struck me as Tom (East) was speaking—and I think it applies to this and to calorimetry and probably to many of the things that we monitor—patient preparation is very important. It's not just a matter of somebody walking in with a device and hooking it onto the endotracheal tube or the ventilator and getting some numbers. You really need a lot more expertise than that.

Pierson: Sure.

Krieger: I want to comment on using a respiratory inductive plethysmograph to measure changes in FRC (or actually changes in thoracic gas volume), which can be done with any external measuring device. If you do use it, you have to have a calibration system that is known to remain stable with changes in patient position, because patients don't lie flat (as we all know). There are a lot of calibration methods that have been used with the old Respitrace system. When we did this study,[1] we used a newer calibration method that is in the commercial Respigraph system, which I will describe in my talk. One of the reasons we developed this calibration technique was so that the thoracic gas volume would remain stable even after patients moved; when we used older calibration techniques, our errors would be prohibitive as far as believing whether or not the external PEEP matched the auto-PEEP.

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Welch: You discussed radiographic techniques, specifically the application of x-rays. Has there been any work done with gated scanners using radionucleotide labelling devices to look at lung volume?

Pierson: I couldn't find any in my literature review.

Lung Mechanics Determinations at the Bedside: Instrumentation and Clinical Application

John J Marini MD

INTRODUCTION

Volume change, the fundamental mechanical event of chest inflation, is driven by pressure gradients and monitored by the measurement of airflow. In a real sense, all indices that characterize the mechanical behavior of the respiratory system derive from just three types of measurement: airway pressure, or inside pressure; intrapleural pressure, or outside pressure; and airflow. If these variables are monitored, other key data of interest can be derived dynamically from intervariable comparisons—eg, alveolar pressure from static airway pressure (at the point of zero flow)—and measured from appropriately timed occlusions of the airway.

Whether generated by muscular or machine power, the total pressure applied to the chest must be dissipated against elastic and frictional forces. Lung expansion is governed by changes in the difference between alveolar pressure (P_{alv}) and intrapleural pressure (P_{pl}), whereas the gradient of pressure from the airway opening to the alveolus ($P_{ao} - P_{alv}$) drives airflow. The elastic forces opposing lung and chest-wall expansion are best assessed under static (or quasi-static) conditions as the compliance of the structure in question. Compliance is defined as the ratio of the system volume (above the equilibrium value) to the pressure required to sustain that volume under static conditions. When the lung is the structure in question and lung compliance (C_L) the measurement of interest, the relevant pressure is alveolar minus intrapleural pressure, or $P_{alv} - P_{pl}$. When the chest wall and its compliance (C_{cw}) is of interest, the relevant pressure can only be assessed under passive conditions as the change in pleural pressure (ΔP_{pl}).

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PRIMARY MEASURES

Airway Pressure

Central airway pressure (P_{aw}) provides information essential for the computing of resistance (R_L) and compliance (C_L) during passive inflation, and for assessing breathing effort during triggered machine cycles. Most clinicians evaluate maximal peak dynamic (P_D) and static, plateau (P_S) pressures but disregard the information contained in the P_{aw} waveform prior to end-inspiration. During passive inflation, these peak pressures are detected with acceptable accuracy by a needle manometer. However, a continuous graphic display of the P_{aw} waveform is necessary for its precise pressure measurement and for capturing information contained in the early portion of a machine-assisted inspiratory cycle.

Because P_{aw} and pulmonary artery pressures vary over a similar range, a pressure transducer (permanent or disposable) identical to that normally used to record pulmonary artery pressure can be used to measure P_{aw} continuously. Filling the transducer with liquid is not necessary. It is important, however, to dedicate a pressure transducer specifically for P_{aw} measurement. (When a single transducer is shared between the pulmonary circulation and the airway, great

Symbols and Abbreviations Used in This Paper

η	— Viscosity
τ	— Time constant
AP	— Auto-PEEP, unexpected (or intrinsic) end-expiratory pressure
C_{cw}	— Chest wall compliance
C_L	— Lung compliance
C_{rs}	— Compliance of respiratory system
C_t	— Thoracic compliance
E_{cw}	— Elastance of the chest wall
E_L	— Elastance of the lung
EMG	— Electromyographic
E_{rs}	— Elastance of the respiratory system
ERV	— Expiratory reserve volume
ET	— Endotracheal
FRC	— Functional residual capacity, or resting lung volume
IC	— Inspiratory capacity
MEP	— Maximal expiratory pressure
MIP	— Maximal inspiratory airway pressure, or P_{max}
MV_T	— Mechanically delivered tidal volume
MVV	— Maximal voluntary ventilation
P_0	— Pressure at zero time
$P_{0.1}$	— Mouth occlusion pressure at 100 ms
$P_{0.1max}$	— Maximal mouth occlusion pressure at 100 ms
P_{alv}	— Alveolar pressure
$P_{alv\text{-}ee}$	— End-expiratory alveolar pressure
$P_{ao} - P_{alv}$	— Gradient of pressure from the airway opening to the alveolus
P_{atm}	— Atmospheric pressure
P_{avg}	— Mean inflation pressure
P_{aw}	— Central airway pressure
P_{cw}	— Average pressure inflating the chest wall
P_{di}	— Transdiaphragmatic pressure
P_{dr}	— Driving pressure
P_D	— Peak dynamic pressures
PEEP	— Positive end-expiratory pressure
P_{es}	— Esophageal pressure
P_L	— Transpulmonary pressure, or $P_{pl} - P_{aw}$
P_{max}	— Maximal inspiratory airway pressures, or MIP
P_{pl}	— Intrapleural pressure
P_{rs}	— Pressure distending entire respiratory system
P_S	— Peak static plateau pressure
P_t	— Recorded pressure at time t
P_T	— Tracheal pressure
P_{tc}	— Pressure at the endotracheal tube connector
PTI	— Pressure-time index
PTG	— Fleisch pneumotachograph
P_{tp}	— Transpulmonary pressure
P_Z	— Pressure at point of inspiratory flow cessation
R	— Resistance (to airflow)
R_E	— Expiratory resistance
R_I	— Inspiratory resistance
RIP	— Respiratory inductive plethysmography
R_L	— Lung resistance
SFT	— Stop-flow technique
T	— Temperature
t_i	— Time of inflation
t_i/t_{tot}	— Duty cycle, or ratio of inspiratory time to total-breathing-cycle time
V	— Volume
$\dot{V}$	— Flow, volume/unit time
V_c	— Compressible gas volume
VC	— Vital capacity
$\dot{V}_E$	— Minute ventilation
$\dot{V}_I$	— Inspiratory flow
V_L	— Lung volume
V_T	— Tidal volume
$V_{T\text{-}c}$	— Tidal volume corrected for gas compression
W_B	— Work per breath
W_I	— Work of chest inflation, or inflation impedance
W_p	— Patient work during machine-assisted inspiration
W_{psv}	— Patient work during pressure support ventilation

potential exists for air embolism to occur with the inadvertent turn of a stopcock.) The tap for temperature measurement located close to the Y-piece serves as a convenient port for sensing central P_{aw}. The bedside monitor and strip chart can then be used to track P_{aw}. To avoid artifacts caused by the kinetic impact of axial flow, the P_{aw} tap should enter the airway perpendicular to the axial stream.

Uses of the P_{aw} Tracing

The *passive* P_{aw} tracing can provide a wealth of useful information. Under conditions of passive inflation, P_{aw} alone can be used to assess overall thoracic impedance (or its subcomponents, R_L and C_L). A 'squared off' inspiratory P_{aw} contour during passive inflation with constant flow may be a clue

to alveolar or airway recruitment and collapse during the tidal volume cycle. Such a squared profile can also mean volume dependence of airway resistance (R_{aw}) or suggest the presence of occult end-expiratory pressure (auto-PEEP, AP). In each of these settings, the patient may benefit from the application of positive end-expiratory pressure (PEEP).

During *active* breathing, the P_{aw} tracing furnishes information of great value in assessing inspiratory effort and the resistance of the mechanical apparatus attached to the patient. The P_{aw} contour of a gently triggered machine-powered breathing cycle should resemble a trapezoid when constant flow is used.[1] Scalloping of the tracing generally indicates vigorous patient effort or, less commonly, inflation impedance that continuously worsens as tidal volume (V_T) increases.

During spontaneous breathing, P_{aw} should ideally be maintained constant at the set value for end-expiratory pressure. The depth and duration of the P_{aw} deflection below the set end-expiratory pressure is inversely proportional to the efficiency of the circuit in maintaining adequate gas flow on demand. Such deflections directly reflect the work done by the patient across the ventilator's external circuitry.

Peak Dynamic Pressure

During passive inflation, P_D indicates the pressure needed to distend the respiratory system at the chosen values for V_T and inspiratory flow ($\dot{V}_I$) (Fig. 1).[2]

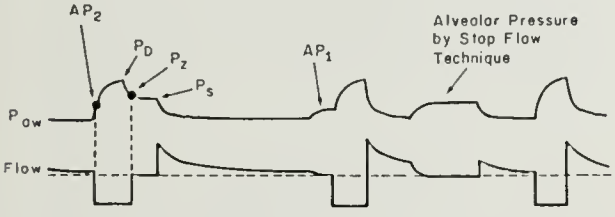

Fig. 1. Simultaneous tracings of airway pressure (P_{aw}) and airflow during controlled volume-cycled ventilation in a patient with airflow obstruction. P_D, P_Z, and P_S represent peak inflation pressures during dynamic conditions, at the point of flow cessation, and after complete equilibration, respectively. Alveolar pressure can be estimated by the stop-flow technique in mid-expiration or at end exhalation (AP, PEEP, $AP1_I$). AP can also be estimated under dynamic conditions from the P_{aw} needed to counterbalance elastic recoil and stop expiratory flow (AP_2). (Reproduced, with permission, from Reference 2.)

P_D is influenced by chest impedance, machine settings, and the resistance of tubing connections located between the pressure tap and the lung. Narrowing of the endotracheal (ET) tube channel by secretions, kinking, or impingement of the tip against the tracheal wall elevates P_D, whereas cuff leaks or ongoing inspiratory effort decreases it. P_{aw} must be sensed beyond the distal (carinal) tip of the ET tube in order for P_D to faithfully reflect the impedance of the lungs and chest wall. Nonetheless, if V_T, $\dot{V}_I$ intensity, and flow pattern remain unchanged, P_D measured under passive conditions can be used to track bronchodilator response.[3] Although tube resistance affects P_D during passive machine cycles, P_{aw} does not reflect ET-tube resistance during spontaneous breathing. The appropriate site for measurement of ET-tube resistance during spontaneous breathing is just beyond the distal (carinal) tip of the endotracheal tube.

Peak Static (Plateau) Pressure

Peak static pressure (P_S) is the sum of end-expiratory alveolar pressure (PEEP or auto-PEEP) and the additional pressure required to distend the respiratory system by the V_T. Changes in C_L and C_{CW} and in thoracic volume influence P_S. P_S is measured at end inspiration under stop-flow conditions by a transient (0.5 to 1.5 second) airway occlusion accomplished manually or by a machine-imposed end-inspiratory pause (Fig. 1).

Post-occlusion, P_{aw} decays rapidly to a stable plateau value over 0.3 to 2.0 seconds as gas redistributes within the lung and stress relaxation occurs. The rapidity with which static equilibrium is achieved depends largely on the duration and homogeneity of inter-regional time constants. For example, patients with chronic obstructive pulmonary disease achieve static equilibrium more slowly than patients with normal lungs. Inability to establish a stable P_S in a passive patient indicates a leak in the ventilator circuitry or lung (eg, a bronchopleural fistula). Values for P_S are generally 1- to 5-cm H_2O lower than the pressure recorded at the exact point of inspiratory flow cessation (P_Z). Therefore, the P_D-P_S difference is likely to be somewhat greater than the actual pressure difference driving inspiratory flow ($P_D - P_Z$).

Under so-called stop-flow, or no-flow, conditions, no pressure dissipates across resistance with flow

stopped. A common system pressure exists, and, consequently, P_S reflects P_{alv}—even though it is measured in the external airway. This same stop-flow technique (SFT) can also be employed to estimate P_{alv} at any other point in the respiratory cycle (Fig. 1). Occlusion at end expiration provides an estimate of end-expiratory alveolar pressure ($P_{alv\text{-}ee}$), a value of great interest in acutely ill patients with airflow obstruction (see AP effect, later in this review).[4]

Assuming that V_T does not change, an increase in P_S may indicate reduced C_{cw} (abdomen or rib cage), reduced C_L, total occlusion of an airway segment, or higher resting lung volume (FRC). P_S remains uninfluenced by secretions, bronchospasm, and other forms of partial occlusion that affect dynamic measurements. FRC variations may also influence the P_S value, both by augmenting the preinspiratory pressure required to initiate $\dot{V}_I$ and by shifting the thorax to a different portion of the pressure-volume curve.

Maximal Inspiratory Airway Pressure

With the airway occluded, isometric fluctuations of central P_{aw} reflect nearly identical changes in P_{pl} and P_{alv}. Therefore, the maximum pressures generated against an occluded airway can help gauge inspiratory and expiratory muscle strength.[5] The force-length properties of skeletal muscle must be understood when one attempts this assessment. For a given level of neural stimulation, the pressure developed by the skeletal muscles of the respiratory system relates directly to precontractile fiber length. Hence, maximal inspiratory airway pressures (MIP, or P_{max}) are developed against an airway occluded at residual volume, whereas maximal expiratory pressure (MEP) is initiated from total lung capacity. (The diaphragm is also more optimally curved at low lung volumes.)

In the intensive care unit, an MIP more negative than −30 cm H_2O is a favorable indicator for weaning.[6] Unfortunately, the MIP index varies widely with patient effort and with thoracic volume at the time of occlusion. A one-way valve that selectively allows expiration ensures that efforts made at the end of an 8- to 10-breath run begin from lung volumes smaller than FRC. When 20 to 25 seconds elapse before releasing the occlusion, *most* patients—even uncooperative ones—are stimulated to high levels of drive.[7]

MIP may be overestimated in patients who are not intubated, because pressure recorded during mouth occlusion can be enhanced by closing the glottis and using the buccal musculature to develop high local suction pressure. In a research setting, this artifact is exposed by deflections of P_{aw} that markedly exceed their esophageal counterparts. For the critically ill patient, intubation eliminates the problem by preventing glottic closure.

The maximal isometric expiratory pressure generated against an occluded airway correlates reasonably well with coughing ability and muscle strength. Unfortunately, the MEP, a value maximized at high lung volumes, demands patient cooperation for a meaningful measurement.

Mouth Occlusion Pressure

Another occlusion pressure, one generated during tidal efforts 100 ms following surreptitious airway occlusion ($P_{0.1}$), provides entirely different information. Although generally regarded as an index of the drive to breathe, $P_{0.1}$ is influenced by muscle strength (and therefore by lung volume). As an isometric measurement, it is not affected by lung impedance.[8,9] During the first 100 to 250 ms of a surreptitiously occluded breath, the subject remains unaware that occlusion has occurred. Therefore, the pressure generated during that interval reflects the output of the ventilatory center during the tidal breathing that preceded the maneuver. For $P_{0.1}$ measurements, P_{pl} can be used as successfully as P_{aw}.

During tidal, machine-assisted breathing cycles triggered from a mechanical ventilator, a delay that approaches or exceeds 100 ms can be imposed by the inspiratory demand valve. This delay effectively occludes the airway for the initial 100 ms of inspiration, during which a fall in circuit pressure results from the tidal patient effort.[10] This pressure drop appears to closely parallel formally assessed measures of $P_{0.1}$.[11] Therefore, when P_{aw} is recorded at a rapid chart speed ($\geqslant$ 50 mm/s), the P_{aw} can often provide an online index of ventilatory drive during triggered machine-assisted breath cycles.

Auto-PEEP

Dynamic hyperinflation results when expiratory time is insufficient to permit deflation to the resting equilibrium position between tidal cycles. Auto-PEEP

(AP), also known as intrinsic or occult PEEP, is a term that refers to P_{alv} when it exceeds P_{aw} at end-exhalation. Detectable AP develops at high levels of ventilation, especially in patients with increased R_{aw}. During inflation with positive pressure, P_{alv} then remains continuously positive, and flow continues throughout exhalation. AP provides important clinical information. The clinical consequences of AP include barotrauma, increased work of breathing, hypotension, and misinterpretation of hemodynamic data. Under conditions of passive exhalation, AP can be measured by occluding the expiratory port of the ventilator circuit just prior to the onset of inspiratory flow.[4] Occlusions initiated too close to end-exhalation underestimate AP by not allowing sufficient time for P_{aw} to reach a plateau value. Conversely, occlusions made too early in the expiratory cycle overestimate AP, because P_{alv} is recorded before the V_T is fully expelled. Some newer ventilators permit accurate measurement of AP by simultaneously occluding the airway at end-exhalation while delaying the subsequent inflation cycle.

AP can also be determined during the initial phase of controlled inflation, provided that exactly simultaneous recordings of flow and P_{aw} are available (Fig. 1).[12] Under totally passive conditions, the P_{aw} at flow onset represents the pressure needed to counterbalance AP and initiate inspiratory flow. Accuracy depends on precise synchronization of flow and pressure measurements, an appropriate pressure scale, and the ability to run the recording paper at high speeds. Recently, it has been suggested that the level of added PEEP necessary to cause a detectable increase in lung volume might also estimate AP,[13] but the estimate, at best, must be considered qualitative.

Esophageal Pressure

Clinical Uses of Pleural Pressure

Knowledge of P_{pl} is crucial in distinguishing the mechanical properties of the lungs from those of the chest wall and in gauging inspiratory effort. Lung inflation is powered during both passive and spontaneous breathing by fluctuations in transpulmonary pressure (P_L, or $P_{pl} - P_{aw}$). The inspiratory change in P_{pl}, the pressure expanding the chest wall under passive conditions, can be used in conjunction with V_T to characterize chest-wall stiffness. Such data enable partitioning of total thoracic impedance into lung and chest-wall components. Finally, the magnitude of P_{pl} determines the pressure surrounding the cardiac chambers and the back pressure to venous return. Some estimate of mean P_{pl} fluctuation is needed for proper interpretation of pulmonary vascular pressure.[14]

Difficulty of Pleural Pressure Estimation

P_{pl} varies from site to site within the thorax, influenced by hydrostatics and geometry. In the upright position, P_{pl} is more negative at the apex than at the base of the lung; P_{pl} varies by $\cong 0.3$ cm H_2O of vertical distance—a difference attributed largely to hydrostatic forces.[15] However, apart from such gravitational effects, local pressures may deviate considerably from the predicted value, due to local deformation of the lung and/or the weight of the mediastinal contents.[15] For example, pressures in the cardiac fossa may exceed those observed elsewhere in the chest because the distended lungs and heart simultaneously expand against one another.[16] In the supine position, a sensor placed behind the heart is influenced by the cardiac weight and therefore records local pressures greater than those obtained in the lateral decubitus position.[17]

Thus, P_{pl}, a surface pressure, is not a unique number, but a quantity whose value is site dependent. A large pneumothorax provides one exception to this general rule, enveloping major portions of the lung in a gas pocket whose pressure is uniform. A large hydrothorax creates uniform pressures at the same hydrostatic level—but accentuates the vertical gradient of P_{pl}. Fortunately, even when the lung is apposed to the chest wall, the P_{pl} tends to *change* more or less uniformly at all points of measurement, so that a single well-placed sensor records average *changes* in P_{pl} with acceptable accuracy.[18]

Because P_{pl} is ordinarily a surface pressure developed in a potential space, any device attempting to measure this surface pressure deforms the lung, altering the recorded value. Multiperforated catheters tend to sense the lowest pressure adjacent to any hole because any higher pressures tend to drive gas or fluid into the catheter lumen toward a point of lower pressure.

Unless there is a generous sheath of gas or liquid to envelope the catheter and provide reliable linkage

to the catheter lumen, strongly positive surface pressures are not sensed precisely by conventional catheter systems (eg, chest tubes). As surface pressure builds within the potential space, soft tissues invaginate the catheter, thereby generating counter-balancing tissue recoil (Fig. 2). Very little compression

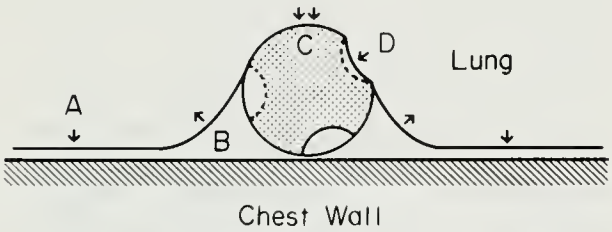

Fig. 2. Cross-sectional view of a multiperforated catheter placed in the pleural cavity. The rounded catheter must displace the deformable lung from the chest wall. Tissue recoil creates regions of increased pressure (C) and (D) and decreased pressure (B) that differ from the true pleural surface pressure P_{pl} (A). Luminal pressure tends to underestimate the true P_{pl} as the recoil of invaginated tissue offsets high local pressures (D). (Reproduced, with permission, from Reference 2.)

of the luminal gas occurs to transmit the surface pressure to the transducer. In similar fashion, strongly negative pressures applied to a drainage catheter (eg, a thoracostomy tube) do not generalize throughout the pleural space. The lung and other soft tissues clog the catheter holes, insulating the majority of the pleural space from the full suction force. It is not surprising, therefore, that pressures more negative than −200 torr have been recorded during chest-tube stripping—without inducing barotrauma.[19]

The Esophagus as a Sampling Site for Pleural Pressure

When air or fluid separate the lung and chest wall, virtually all types of intrathoracic-pressure sensors are equally accurate. However, the same is not true when the visceral and parietal pleural surfaces remain in contact. In the experimental setting, flat, flexible balloon sensors that conform to the contours of the lung and chest wall record surface pressure best.[20] Cylindrical catheters are less desirable. Although such balloon wafers can only be placed surgically, the flaccid esophagus provides convenient access to the pleural boundary. Esophageal pressure (P_{es}) can be transduced by a liquid filled, open-ended catheter, by a transducer-tipped catheter, or, most commonly, by a balloon-tipped catheter. A balloon catheter introduced into the lower or middle third of the esophagus senses local pleural pressure accurately, provided the catheter does not deform the esophagus sufficiently to trigger smooth muscle contraction. Pressures within the upper third of the esophagus may be influenced by adjacent tracheal pressure or esophageal opening artifacts.[18,21] In the upright or lateral decubitus positions, the local pressure sensed by a well-placed esophageal balloon estimates global P_{pl} with acceptable accuracy and tracks pressure fluctuations faithfully. In the supine position, however, the mediastinal contents weigh on the inferiorly positioned esophagus and balloon, and the catheter is influenced by a higher-than-average hydrostatic pressure.[18] These effects raise the end-expiratory P_{es}, which then fails to reflect the global value of interest. Whatever the inaccuracies of the absolute P_{es} value may be, fluctuations in P_{es} and P_{pl} may track together acceptably well.

Balloon Construction

Esophageal balloons are usually constructed of thin latex rubber tied over a length of polyethylene catheter stock.[22] Multiple perforations in the catheter are arranged in a spiral fashion. The balloon is about 10-cm long, a dimension great enough to sample a sizeable segment of the esophagus. Balloons to measure gastric pressure are made of similar materials, but are usually 5-cm long. Typically, the gastric balloon is filled with 5 mL of air to encourage flotation on the surface of the gastric contents.

Until quite recently, a multilumen catheter device has been commercially available that combines the functions of a nasogastric sump tube and an esophageal balloon. Although the caliber of this tube is much greater than that of a standard esophageal balloon catheter, fluctuations in pleural pressure appear to be tracked acceptably well.[23] Unfortunately, the future availability of this instrument appears to be in serious doubt.

Positioning of the Esophageal Balloon

The balloon is then withdrawn from the stomach approximately 10 cm to the point at which the

inspiratory deflections first become negative, signalling the entry of the top of the balloon into the thoracic cavity. This places the main body of the catheter in the lower third of the esophagus. The lowermost portion of the balloon should rest ≅ 40 cm from the nares at that point. The balloon is first filled with 6 to 8 mL of air and then emptied of all but the last 0.5 to 1.5 mL. Some authors have suggested the use of only 0.1 to 0.2 mL of air; however, when P_{es} is measured in mechanically ventilated patients, it is important to leave enough residual air for the balloon to remain sensitive despite the compressive effects of positive pressure. Several sets of guidelines have been developed for achieving optimal P_{es}-balloon placement. Most methods advocate initial passage of the air-primed balloon into the stomach, detected by positive-pressure deflections during rapid, forceful inhalations (sniff maneuver).[22,24] The balloon is then withdrawn to the point where the inspiratory pressure deflections first become negative, signalling entry of the top of the balloon into the thoracic cavity. (As noted earlier, multi-perforated catheters tend to transmit the lowest pressure that exists anywhere along their length.)

Final positioning of the balloon can be confirmed by comparing the negative-pressure deflections of P_{es} and P_{aw} during inspiratory efforts made against an occluded airway (ΔP_{es} and ΔP_{aw}).[24] During occlusions, no significant change in lung volume occurs, so that changes in average transpulmonary pressure [ΔP_L, or $\Delta(P_{aw} - P_{es})$] should also be negligible. A well-positioned balloon would therefore yield changes in P_{es} that are nearly equivalent to simultaneously measured changes in P_{aw}. In any posture, a site can usually be found where fluctuations in P_{aw} and P_{es} are very similar during occluded efforts. Agreement tends to be least, however, in the supine position; therefore, P_{es} should be measured in the upright or in the lateral decubitus positions whenever feasible. Optimal placement of the balloon during passive inflation (no spontaneous effort) is very difficult. As a general rule, the tip of the esophageal balloon catheter should be placed 40 to 45 cm from the nose.

P_{es} can be used in many derived physiologic measurements of clinical interest. During spontaneous breathing, esophageal manometry is the only technique that provides a way of quantifying effort as well as the C_L and R_L. P_{es} can also be used to track dynamic hyperinflation, the difference between end-expiratory P_{es} and the P_{es} corresponding to zero flow quantifies the counterbalancing pressure needed to overcome deflation recoil and initiate inspiratory airflow. During passive inflation, ΔP_{es}, used in conjunction with V_T and ΔP_{aw}, enables computation of the relative compliances of the lung and chest wall. Knowledge of P_{pl} also facilitates interpretation of central venous and pulmonary vascular pressures.

Flow

Utility of Measurement

Knowing the rate of airflow enables one to calculate resistance (R) from the quotient of driving pressure (P_{dr}, ΔP_L, or $P_{ao} - P_{alv}$) and flow ($\dot{V}$);

$$R = \frac{\Delta P}{\dot{V}}.$$

During passive ventilation with constant V_T and system compliance, changes in inspiratory resistance manifest as changes in P_{aw} when $\dot{V}$ is constant (ie, volume-cycled ventilation) or as changes in the flow profile when P_{ao} remains constant (eg, pressure-controlled ventilation). Integrating flow with respect to time yields inspired or expired volume, an essential measurement for the computation of C_L and for monitoring the adequacy of tidal breathing.

The flow tracing can be used for several diagnostic purposes distinct from routine calculations of inspiratory resistance (Fig. 3). For example, flow can

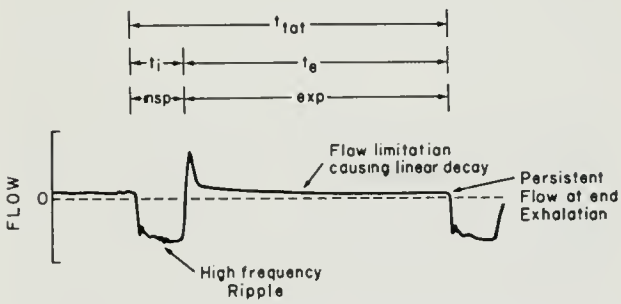

Fig. 3. Flow tracing during controlled volume-cycled ventilation with constant flow. Inspiratory ripple indicates turbulence. Flattened (rather than exponential) expiratory profile and persistence of flow at end exhalation are features of flow limitation and hyperinflation, respectively. (Reproduced, with permission, from Reference 2.)

be used in conjunction with simultaneous estimates of P_{alv} (obtained by the stop-flow technique) to calculate expiratory resistance.[25] The flow tracing suggests expiratory flow limitation when its profile appears to decay in a linear, rather than clearly exponential, fashion. Another clue to flow limitation is a post-occlusion 'spike' of flow that appears before flow limitation is re-established. Flow limitation can be confirmed by observing negligible change in the flow profile despite the addition of a supplemental resistor to the expiratory line.[3] Persistence of significant flow at end exhalation indicates failure of the respiratory system to achieve its resting equilibrium position (hyperinflation, AP).[4,12] Finally, high-frequency ripples on the inspiratory flow waveform indicate turbulence and the potential need to clear secretions or water condensate accumulated in ventilator tubing.[26]

Technique of Measurement

The majority of techniques for measuring airflow perturb the airstream in some fashion, using its physical properties to generate signals proportional to the rate of bulk gas transfer. Kinetic energy can be transformed to rotational movement (rotameter) or dissipated through a laminar resistance (Fleisch pneumotachograph, PTG). Boundary currents can cool a warmer object (filament or hot-wire anemometer), and flow-related turbulence can be utilized to disrupt the transmission of an orthogonal ultrasonic beam (vortex-shedding flowmeter). Flowmeters (anemometers) can be functionally categorized as pressure-based and nonpressure-based devices.

Pressure-Based Flowmeters

Pressure-based flowmeters interpose a fixed (unchanging) resistance into the airstream. The pressure gradient developed across this resistor is proportional to flow, so long as flow remains laminar.

Establishing laminar flow. Laminar flow through these devices is achieved by reducing the factors that increase turbulence: high velocity, luminal irregularities, and sudden changes in cross-sectional area.[27] Thus, laminar flow is favored by increasing the flowmeter's diameter and by adding gently tapered cones and long smooth-bore tubing to ensure a sufficient "entrance length" for the establishment of laminar conditions.[27] The ideal proportion for a smooth-bore tube attachment is a length five times its width, with a diameter equal to that of the flowmeter. Bi-directional flow measurements require these tubes to be placed on both sides of the PTG. Unfortunately, these extensions increase dead space when placed between the airway opening (endotracheal tube) and the Y-junction of inspiratory and expiratory tubing. Wide-caliber flowmeters maintain laminar conditions and accurate flow measurement at greater velocities. However, such PTGs are relatively insensitive to flows in the lower range and interpose high dead-space volumes. The smallest PTG that maintains laminarity for the flows to be measured should be chosen.

Maintaining constant resistance. When gas flows under laminar conditions, resistance is altered by gas viscosity (η) and the length (l) and radius (r) of the conduit, as defined by Poiseuille's law ($\dot{V} = \Delta P (8(\eta)l/r^4)$,[28] because resistance = $\Delta P/\dot{V}$ and $R = r^4/8(\eta)l$. Unless mucus plugging or condensation occur within the PTG, l and r are unchanging characteristics of the flowmeter itself. In the clinical setting, moisture can exert dramatic effects on flowmeter resistance and hence on flow measurement.

Warming the PTG to body temperature helps decrease condensation and limits accumulation of moisture in the instrument.[29] Although viscosity remains unaffected by gas density, η is influenced by the elemental composition of the gas and independently by its water content and temperature.[30] After calibration, alterations in these properties necessitate use of a correction factor to obtain accurate measurements.

Types of pressure-based flowmeters. Like all pressure-based flowmeters, the Fleisch PTG records flow by sensing the pressure developed across a small fixed resistance. A collection of warmed metallic capillary tubes bound together in a cylinder provide this resistance while maintaining laminar flow.[31] Even when heated, condensation sometimes collects in the flowmeter during extended usage.

Screen-type PTGs reduce dead space and improve frequency response by substituting a metallic mesh for the capillary network of the Fleisch unit.[31]

However, because these screen-type instruments frequently clog with moisture, regular cleaning is required to minimize artifact. Disposable fiber screens minimize or eliminate the need for cleaning but require frequent changes because of moisture absorption. As a further drawback, such units permit only unidirectional flow measurement.

Compensating for extremes in flow. Uncompensated, pressure-based flowmeters maintain accuracy only over the flow range that corresponds to laminar conditions. Some flowmeters are designed to compensate for flows beyond the limits of the designated range. Adjustment (linearization of flow) can be accomplished electronically or mechanically. Digital microprocessors can be used to achieve electronic linearization. During the calibration process, a computer assigns known flow inputs to the pressures developed across the PTG. In use, the preprogrammed microprocessor converts the transduced pressure signal from the PTG into a compensated analog signal corresponding to actual flow.

Variable-orifice PTGs compensate mechanically for flows outside the laminar range of most Fleisch units.[32] Such devices resemble a hollow cylinder interrupted by a perforated disc. Pressure is tapped within the cylinder on both sides of the disc. During flow, the disc's aperture (the fixed portion of the orifice) creates a detectable pressure drop, yet is sufficiently large to allow passage of small water droplets without condensation. The turbulent flow produced at the orifice would cause the pressure developed across a fixed resistor to increase as a power function of flow. However, an elastic flap positioned over the orifice (and displaced by unidirectional flow) enlarges the effective orifice in proportion to flow, compensating for the potential effects of turbulence on the resistance calculation. Hence, the resistance (and pressure drop) across the total orifice remains mechanically linearized to flow.

Non-Pressure-Based Flowmeters

Turbine. Turbine flowmeters (eg, the Wright Respirometer) use the 'windmill' principle. Flowing gas spins a turbine geared to a needle gauge. Flow need not be laminar and water content is not a major concern. These turbine units are lightweight and relatively inexpensive, but lack precision.[33] Rotational inertia and acquired momentum of the mechanical unit diminish responsiveness. At flow onset, these units are relatively slow to respond; and, once spinning, they continue to register even after flow cessation. Newer devices convert the turbine spin electronically to numerical values, without linkage to a physical gauge, helping to decrease the impact of mechanical inertance on the flow measurement.

Vortex shedding. Vortex-shedding PTGs use struts in a laminarized airstream to disrupt streamlines and produce turbulence.[27] Turbulent swirls of air are termed vortices. A sensor detects an ultrasonic beam projected across the tube, orthogonal to its main axis. Vortex-caused interruptions of the beam give rise to electrical pulses that are quantified and correlated to flow. Because this technique relies on the creation of turbulence, very low flows may not disrupt laminarity sufficiently to register. Conversely, very high flows may create so much turbulence that vortices coalesce, reducing the extent of beam interruption and leading to falsely low flow values. Vortex-shedding flowmeters impose little dead space, use few moving parts, and are relatively insensitive to changes in gas composition. Although not greatly affected by humidity, water condensed on the struts or sensors can lead to erroneous values—a common problem in the mechanically ventilated patient.

Non-vortex-shedding ultrasonic flowmeters. Sound waves can be projected along the longitudinal axis, parallel to flow, rather than across it.[34] The speed of ultrasonic wave transmission can be helped or hindered by the air current. Ultrasonic flowmeters pulse waves along the axis of flow; differences in transmission velocity reflect changes in airflow. Moisture and viscosity do not alter the accuracy of these bi-directional anemometers.

Hot-wire anemometers. Hot-wire anemometers position a thin heated filament in the airstream. The amount of additional current required to maintain the wire's temperature is proportional to flow. The precision of measurement is affected by ambient temperature, altitude, moisture, gas density, and turbulence.

Volume

Bedside determinations of volume made in the intensive care unit include absolute measurements

(FRC and dead space) and those volumes measured relative to FRC: V_T, vital capacity (VC), and the volume trapped in association with AP.

Volumes Relative to FRC

Utility of measuring volumes relative to FRC—*Tidal Volume.* Shallow tidal volumes ($<$ 4 mL/kg) suggest major limitations of strength or ventilatory drive. Measurements of V_T also permit the calculation of minute ventilation ($\dot{V}_E$), a key indicator of the work of breathing, and maximal voluntary ventilation (MVV), an index of ventilatory reserve and endurance. In the intubated patient, MVV and $\dot{V}_E$ can predict the outcome of a weaning trial. A resting $\dot{V}_E < 10$ L/min coupled with a MVV $\geq 2\ \dot{V}_E$ usually indicates a breathing reserve adequate for withdrawal of support.[6]

Comparisons of inspiratory and expiratory V_T permit detection of gas leakage in the patient-ventilator system. Inspiratory volumes that substantially exceed expiratory volumes on each tidal breath suggest air leakage around the ET-tube cuff or through a tubing connector or bronchopleural fistula.

Thoracic compliance (C_t) can be determined mathematically using V_T corrected for gas compression ($V_{T\text{-}c}$) and two specific airway pressures (P_S and end-expiratory alveolar pressure, $P_{alv\text{-}ee}$); $C_t = V_{T\text{-}c}/(P_S - P_{alv\text{-}ee})$.[35] As will be detailed later, changes in C_t reveal disease processes, loss or recruitment of lung volume, iatrogenic complications (pneumothorax, mainstem-bronchus intubation), or the impact of therapeutic interventions.

Utility of measuring volumes relative to FRC—*Vital capacity.* As an integrative measure of thoracic elastance, volitional effort, and muscle strength, vital capacity (VC) is a valuable indicator of weaning capability in cooperative patients, with values exceeding 10 mL/kg portending a favorable outcome.[36] Although VC is normally considered a measurement that requires full cooperation, much of the information it provides regarding chest mechanics can be ascertained in poorly cooperative subjects by nonconventional methods. The cumulation of inspired volume during passive inflation to a fixed high airway pressure[37] and breath-stacking using a one-way valve can both yield accurate estimates of inspiratory capacity in a poorly cooperative patient. Although neither of these involuntary methods is a reliable index of strength, both test the elastic properties of the system.

Utility of measuring volumes relative to FRC—*Volumes associated with AP.* If the patient remains passive, dynamic hyperinflation can be quantified by allowing the lung to decompress fully to its equilibrium position after a routine tidal inflation. The extent of end-expiratory lung expansion above the point of lung-chest wall equilibrium is simply the difference between total exhaled volume (to zero flow) and V_T.[38] This volume can also be estimated from the product of C_t and AP.

Technique of Measuring Volumes Relative to FRC

Importance of gas compression. All tidal volumes measured during positive-pressure ventilation are affected by gas compression and circuit distensibility. Compressible volume does not contribute to alveolar ventilation. If compressible gas volume (V_c) is not considered when setting machine-delivered V_T, the patient may receive a considerably smaller delivered volume than anticipated. Although its importance is well recognized in infants, V_c is seldom calculated precisely in adults. Yet, compressible losses influence gas exchange, as well as all calculations of thoracic mechanics (eg, compliance) that depend on V_T, especially at high cycling pressures. (Pediatric ventilator circuits are intentionally constructed with minimal internal volume and noncompliant tubing to minimize gas storage effects, which would otherwise prove overwhelming in relation to small infant V_Ts.)

Factors that influence V_c include not only the peak gas pressure generated, but also the pliability, cross-sectional area and length of circuit tubing, humidifier size and liquid volume, and the presence of other gas chambers within the ventilator itself. In addition, some types of ventilators have high internal compressible volumes. The compression ratio: CR = V_c:P_D, an expression of compressible volume per unit of cycling pressure, is not constant but varies in curvilinear fashion with P_D. Thus, with most newer machines, little gas volume is compressed at low cycling pressures ($<$ 15-cm H_2O), whereas major gas storage occurs at very high dynamic pressures ($>$ 50-cm H_2O). Depending on the specific ventilator, the

type of circuit tubing, and the cycling pressure, CR can range from as little as 0.3 to as much as 5 mL/cm H_2O.[39]

Considerations of compressible volume can prove of crucial importance, influencing the apparent power of the ventilator. For any V_T setting, actual ventilatory efficiency falls as C_L worsens. Some of the more recently developed ventilators (1990) automatically evaluate and compensate for the V_c in their displays of V_T. In nonautomated systems, compensation for V_c is achieved by subtracting an estimate of V_c from the total measured V_T or by measuring exhaled volume mouthward of the ventilator's Y-piece.

Methods of Measurement

Volumes measured relative to FRC can be determined by mechanical spirometry, by integration of airflow (so-called electronic spirometry), or by tracking movements of the chest wall and abdomen (rather than movements of respired gases).

Airway-dependent volume monitoring. Mechanical spirometers (water-sealed, dry rolling-seal, wedge, bellows, and turbine) have limited use in the ventilated patient, largely because they are inconvenient to use and do not lend themselves to continuous volume measurement. Furthermore, inertial properties may compromise technical accuracy when breathing is performed at rapid frequencies or high flows.[29] V_T can be accurately assessed by electronic integration of measured flow. Most errors in measurement are secondary to inaccurate flow signals, baseline drift, or alterations in temperature. A drift check can be done by providing a zero flow signal and observing the recorded output. Cumulation of volume under no-flow conditions indicates the presence of an electronic bias that must be counterbalanced by adjusting an electronic offset.

Pressure-based PTGs require differential sensing across an imposed resistance to airflow. A small leak or condensation in one of the pressure taps leading to the transducer generates a differential pressure across the sensitive transducer membrane. Such problems can produce a consistent bias pressure across the transducer (and a resulting inspiratory or expiratory drift) whenever the airway is pressurized, even during periods when no gas is flowing (eg, PEEP). The presence of mechanical drift is confirmed by introducing a known volume through the instrument from both directions. Large differences produced in the volume measurement suggest asymmetrical condensation of the PTG-transducer tubing or a leak in the connections to the flow-measuring pressure transducer.

As predicted by Charles' gas law ($V_1/T_1 = V_2/T_2$), all airway-invasive methods of measuring volume (V) are affected by differences in temperature (T) that exist between the patient and the point of measurement. Volume measurements made at temperatures differing from body conditions require a temperature-related correction factor that accounts for difference in dry gas volume and water vapor pressure.[27] Flowmeters placed close to the proximal end of the ET tube obviate the need for temperature correction.

Non-airway-dependent volume monitoring. Fluctuations in uni-dimensional and bi-dimensional measures of the thoracoabdominal shape (diameter and circumference) correlate with volume changes.[40] Changes in compartmental volume can be monitored noninvasively with devices that apply principles of fluid mechanics or electromagnetics.

Mechanical strain gauges[41] respond to volume changes by registering the pressures generated within mercury- or air-filled rubber tubes surrounding the chest wall. These instruments are cumbersome, inherently inaccurate, and seldom used because they sample changes in only a single plane of one thoracic compartment.

Volume changes give rise to alterations in the electrical impedance of the thorax. When a constant high-frequency current is applied, impedance can be measured continuously by surface electrodes (optimally placed along the fifth intercostal space).[42] Spontaneous movement, postural shifting, and diaphoresis create such large inaccuracies that this methodology is not clinically useful for quantitative volume measurements. (However, because this technology reflects qualitative directional changes in thoracic volume, it does distinguish inspiration from expiration, and thus determines the duty cycle or ratio of inspiratory time to total breathing cycle time, t_i/t_{tot}.)

Magnetometers use volume changes to vary the intensity of the current induced by a magnetic field.[43,44] Parallel coils are placed anterior and

posterior to the chest and abdomen. An electrical current is passed through one coil, creating a magnetic field that induces a current in the opposite coil. The intensity of the induced current varies with changes in thoracic and abdominal diameters. Magnetometers are unwieldy and require frequent calibration, limiting their value in the clinical setting.

Respiratory impedance (inductance) plethysmography (RIP) has become a popular clinical tool to measure volume and breathing pattern. Wide elastic bands enclosing teflon-insulated electrical coils are placed around the chest and abdomen. Expansion of the rib cage and abdomen alter the inductance, changing the oscillator-generated frequency of the electrical current coursing through them.[41] Of the available non-airway-dependent monitors, RIP is least affected by changes in body position, provided the bands fit snugly and are not displaced. Of the multiple calibration techniques available, the least-square technique is perhaps the most accurate and easiest to perform.[45,46]

Changes in FRC. Although repeated determinations of FRC by gas dilution are impractical, changes in FRC can be approximated easily, noninvasively, and with a precision acceptable for most clinical purposes by direct-current-coupled impedance plethysmography.[47] When tidal movements of the chest and abdomen are synchronous, the end-expiratory position of the summed tracing varies semiquantitatively with FRC, essentially providing a spirometric tracing without connection to the airway. When thoracoabdominal movements are asynchronous or paradoxical, the summation signal continues to reflect overall changes in FRC.[48] Furthermore, its component tracings of rib-cage and abdominal motion indicate distributional shifts of volume that go undetected by gas dilution techniques.

A simple one-way valve can be used in poorly cooperative patients to determine inspiratory capacity (IC) and expiratory reserve volume (ERV) from the stacked tidal efforts of eight to ten consecutive breaths[49] (Fig. 4). Such maneuvers can be stressful to critically ill patients and therefore must only be attempted with appropriate caution. Preoxygenation with multiple breaths of oxygen should precede the stacked ERV maneuver, during which the patient is deprived of fresh gas for 20 to 25 seconds. (Of course, when the stacked IC maneuver is performed with 100% oxygen, preoxygenation is not indicated.)

SECONDARY MEASURES

Having reviewed the primary measurements needed to evaluate lung mechanics—airway and esophageal pressures, flow, and volume—I will now focus on their practical application in derived indices of mechanics (resistance, compliance, impedance), energetics (work, pressure-time product), and capability (drive, muscle strength, endurance).

Mechanical Demands

Elastance and Compliance

Definitions. Elastic properties can be characterized by the relationship between volume and the static pressure required to maintain it. For the normal lungs and chest wall, changes in pressure (ΔP) and volume (ΔV) bear a more or less linear relationship through most of the physiologic range, so that a single number, elastance = $\Delta P/\Delta V$ (or its reciprocal, compliance = $\Delta V/\Delta P$), serves to characterize this relationship. For the passive respiratory system, the distending pressure across the lungs is the difference between alveolar and pleural pressures ($P_{alv} - P_{pl}$), and for the passive chest wall, the gradient is pleural pressure – atmospheric pressure (P_{atm}), $P_{pl} - P_{atm}$ (Fig. 5). Thus, the static pressure distending the entire respiratory system under passive conditions can be expressed:

$$P_{rs} = (P_{alv} - P_{pl}) + (P_{pl} - P_{atm}) = P_{alv} - P_{atm} = P_{alv}.$$

Knowledge of pleural pressure (ie, esophageal pressure, P_{es}) allows partitioning of the P_{aw}-P_{atm} gradient and determination of the individual compliances of the chest wall and lung. Because the elastances of two structures arranged in series are additive, the elastance of the respiratory system (E_{rs}) is the sum of its lung (E_L) and chest-wall (E_{cw})

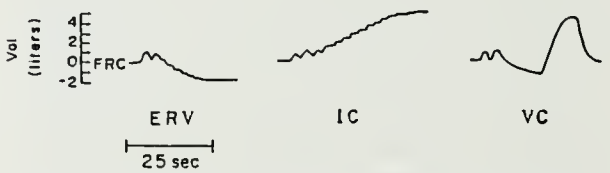

Fig. 4. Comparison of a standard VC maneuver with estimates of its major components—ERV and IC obtained during tidal efforts stacked by one-way valving. (Reproduced, with permission, from Reference 2.)

components: $E_{rs} = E_L + E_{cw}$.[51] Expressed in terms of compliance (the reciprocal of elastance), the equivalent expression becomes:

$$\frac{1}{C_{rs}} = \frac{1}{C_L} + \frac{1}{C_{cw}},$$

and

$$C_{rs} = C_L \frac{(C_{cw})}{(C_L + C_{cw})}.$$

Caution must be exercised when interpreting compliance at the bedside because this measure is sensitive to absolute lung size as well as to volume change. Certain patients who lose absolute resting lung volume (V_L) (eg, with pneumonectomy or massive atelectasis) will have lower measured compliance, but unchanged distensibility of the remaining lung tissue. This problem can be resolved by computing specific compliance. (Specific compliance = measured compliance/absolute lung volume).[52] However desirable it may be to compute C_{rs}/V_L when evaluating tissue elastic properties, it is very difficult to determine absolute lung volume in mechanically ventilated patients.

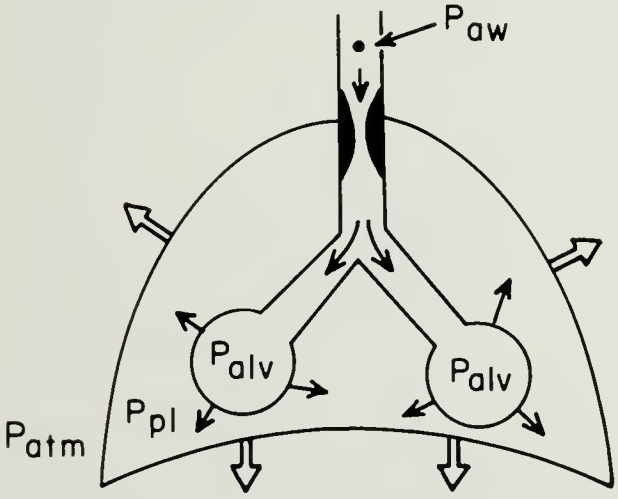

Fig. 5. During mechanical ventilation under passive conditions, the difference between airway pressure (P_{aw}) and atmospheric pressure (P_{atm}) is the gradient driving flow and expanding the respiratory system. This pressure gradient is dissipated in resistive work ($P_{aw} - P_{alv}$) × Vdt and in the elastic work needed to expand the lungs ($P_{alv} - P_{pl}$) × Vdt and chest wall ($P_{pl} - P_{atm}$) × Vdt. Total work performed is quantified by ($P_{aw} - P_{atm}$) × Vdt. (Reproduced, with permission, from Reference 50.)

In a perfectly elastic system, the distending pressure-volume relationship remains linear at all volumes, and its slope does not depend on the phase of respiration. The pressure-volume relationship of the respiratory system, however, is often curvilinear and sometimes demonstrates marked hysteresis (ie, the shape and/or slope of the pressure-volume curve varies between the inspiratory and expiratory half cycles, Fig. 6).[53] Such hysteresis is accentuated when

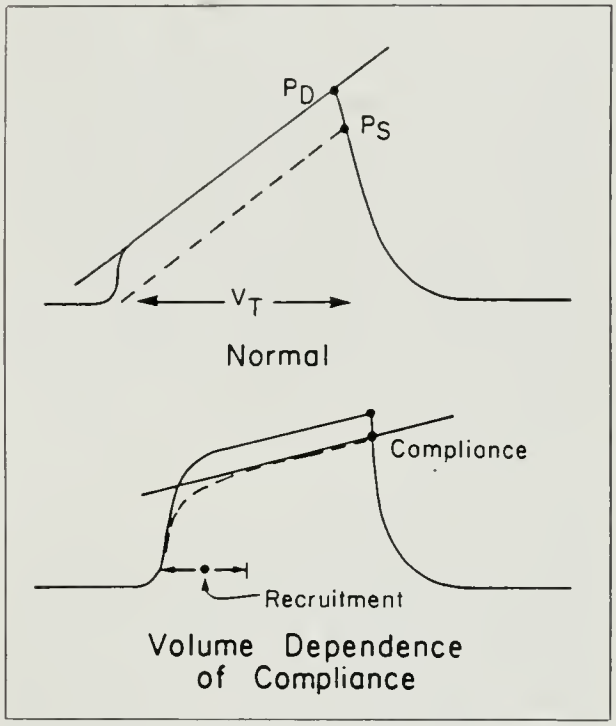

Fig. 6. Airway (——) and alveolar (----) pressures during passive inflation with constant flow. Compliance is nearly constant over the tidal range in normal subjects (A) as shown by the constant slope of the airway pressure-volume relationship. However, in many critically ill patients, hysteresis may cause the alveolar pressure-volume relationship to be curvilinear so that compliance improves as alveoli are recruited at the beginning of the tidal breath (B). During exhalation, alveolar units are derecruited. (Reproduced, with permission, from Reference 50.)

there is tidal recruitment and derecruitment of alveolar units. Despite its potential value in clinical decision making, characterization of hysteresis (and its response to PEEP) is seldom performed in the clinical setting. Instead, the slope of the line joining the zero-flow points of end-expiration and end-inspiration is usually reported, and a single value for compliance calculated.

C_{rs} can be altered by any process that stiffens the lung or chest wall. The apparent stiffness of the lung is increased by hyperinflation, pulmonary infiltration, and volume loss, whereas collections of air or fluid in the pleural space, burn eschar, obesity, ascites, muscular effort, and position changes increase apparent chest-wall stiffness.

Utility of Compliance Calculation

C_{rs} has many potential applications in the ventilated patient. For example, the course of infiltrative disease can be tracked with serial measurements.[54] C_{rs} can also be used to monitor and modify such therapeutic interventions as V_T and PEEP, diuresis, paralysis, and positioning.[55] C_{rs} improves as PEEP recruits alveoli. However, after full recruitment, further increments of PEEP may increase dead space and reduce C_{rs} as overdistended lung approaches its elastic limit. Paralysis often improves the functional compliance of the chest wall. Position-related changes in the geometry of the respiratory system may profoundly affect C_{rs}.

Measurement of Compliance

C_{rs} can be determined by measuring the peak static (P_S) and end-expiratory alveolar pressures ($P_{alv\text{-}ee}$) in conjunction with the volume delivered during a passive tidal inflation.[56] In these calculations, the volume actually delivered to the patient can be measured by a pneumotachograph attached directly to the ET tube or calculated by subtracting an estimate of the amount of gas trapped in the compressible tubing from the expelled V_T (MV_T). (As already noted, some of the newer ventilators automatically adjust for gas compression in their V_T readouts.) Calculations of the externally stored or trapped volume ($P_D \times$ circuit compliance factor) are only estimates, as the true circuit compliance factor varies with dynamic pressure.[39] In the presence of auto-PEEP (AP) or added PEEP, the change in pressure distending the respiratory system during tidal inflation is $P_S - P_{alv\text{-}ee}$, where $P_{alv\text{-}ee}$ = AP or PEEP.[12] Hence, compliance of the respiratory system is mathematically defined as:[35]

$$C_{rs} = \frac{[MV_T - (P_D \times \text{circuit compliance factor})]}{(P_S - P_{alv\text{-}ee})}.$$

When the patient makes spontaneous efforts, C_{cw} (and therefore C_{rs}) cannot be determined because the effective distending pressure of the chest cannot be measured when the respiratory muscles are active. C_L, however, can be computed if P_{es} and flow (and V_T) are monitored, enabling transpulmonary pressure ($P_{alv} - P_{es}$) to be estimated under quasi-static (zero-flow) conditions.[57]

Resistance

Utility of the resistance calculation. Resistance (R), the frictional component of inflation impedance, is defined as the ratio of driving pressure (P_{dr}) to the resulting flow:

$$R = P_{dr}/\dot{V}.$$

Although the majority of computed resistance arises in the airway passages, clinical calculations of resistance (as defined by the just given formula) include much smaller components of tissue resistance and system inertance. Resistance exhibits volume and flow dependency (Fig. 7).[58] Airway tethering and

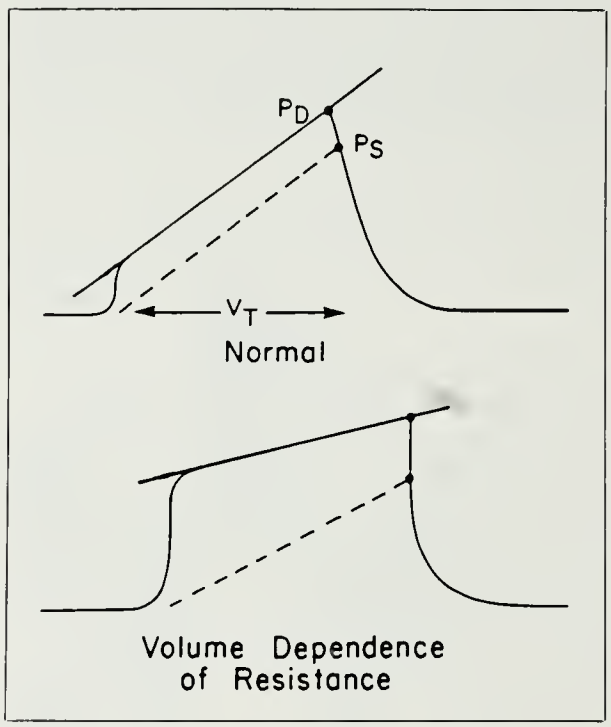

Fig. 7. Airway (solid line) and alveolar (dashed line) pressures during passive inflation with constant flow. Resistance across the respiratory system is fairly constant in normal subjects, as reflected in the unchanging P_{aw}-P_{alv} difference (A). In many patients, however, resistance is volume-dependent over the tidal range, as exemplified by the wider difference between airway and alveolar pressures at the onset of the inflation cycle (B). (Reproduced, with permission, from Reference 50.)

luminal diameter are proportional to the absolute lung volume, especially at volumes below FRC. In patients with advanced degrees of airflow obstruction, resistance is significantly volume-dependent throughout the vital capacity range.

For the latter patients, inspiratory and expiratory resistance (R_I and R_E) often differ. R_E increases dramatically over the course of a tidal deflation in those with dynamic airway collapse during tidal breathing.[59,60]

Measurement of inspiratory resistance. During passive mechanical ventilation with square-wave flow, R_I at end-inspiration can be estimated by dividing the difference between P_D and P_S by the machine-delivered flow.[61] This calculation of R_I characterizes the frictional impedance of spontaneous breaths, to the extent that resistance remains constant during inflation and that the V_T and flow range are representative of the spontaneous values. (As noted earlier, the Δ [$P_D - P_S$] includes a nonfrictional component related to stress relaxation and redistribution of lung volume during the hold period.)

An alternative method for estimating P_S that does not require an end-inspiratory hold is to measure the zero-flow pressure (P_Z) at end-inspiration (Fig. 8). This measurement requires simultaneous, high-speed (50 mm/s) recordings of flow and pressure. Because

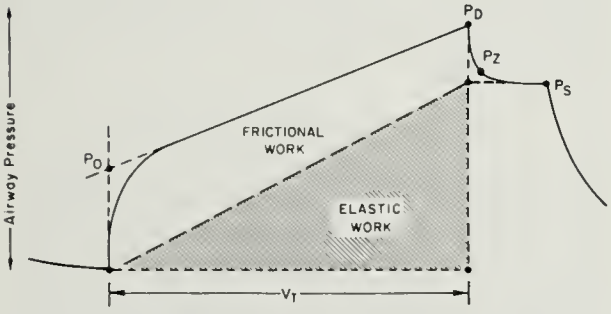

Fig. 8. Under passive conditions, inspiratory resistance (R_I) can be calculated from the airway pressure tracing (P_{aw}) generated by constant flow in three different ways: (1) $R_I = (P_D - P_S)/\dot{V}$, where P_D = peak dynamic pressure, P_S = peak static (plateau) pressure, and $\dot{V}$ = inspiratory flow. (2) Substituting P_Z (P_{aw} measured at the zero-flow point of end-inspiration) for P_S eliminates the need for a stop-flow measurement: $R_I = (P_D - P_Z)/\dot{V}$. (3) The pressure at the onset of inspiration (P_0) is approximated by extrapolating the tangent to the "ramp" of the P_{aw} curve to time zero; $R_I = (P_0 - P_{alv\text{-}ee})/\dot{V}$, where $P_{alv\text{-}ee}$ equals the alveolar pressure at end-exhalation. (Reproduced, with permission, from Reference 50.)

P_Z usually exceeds P_S by 10 to 20% of the P_D-P_S difference, 'resistances' calculated using $P_D - P_Z$ will be smaller than those computed using the P_D-P_S difference.[62] As recent work suggests,[63] the P_Z-based estimate may respond differently to volume and flow in normal subjects than does the P_S-based estimate. However, this requires confirmation in the clinical setting. According to this interpretation, P_S-based (R_I) estimates incorporate tissue as well as airway resistance. Whereas, P_Z estimates are more heavily influenced by airway resistance alone.

During ventilation under passive conditions with square-wave flow, R_I can theoretically be estimated by extrapolating the ramp of the P_{aw} tracing to flow onset (pressure at zero time, P_0).[64,65] The elevation of P_0 above the end-expiratory alveolar pressure baseline ($P_{alv\text{-}ee}$) defines the pressure gradient existing between the central airway and the alveoli at FRC. Therefore, when the pressure gradient at flow onset ($P_0 - P_{alv\text{-}ee}$) is divided by $\dot{V}$, an estimate of R_I near FRC is obtained. Unfortunately, this methodology is fraught with potential problems,[62] yielding inaccurate results whenever (1) the P_{aw} tracing lacks a defined linear segment, (2) AP is present but not accounted for (exaggerating the resistive pressure gradient), (3) lung units are recruited early in inspiration, (4) inspiratory inertia is disproportionately large early or late in the cycle, or (5) overdistention late in the tidal inflation steepens the pressure slope.

Measurement of expiratory resistance. Simultaneous estimates of P_{alv}, of pressure at the endotracheal tube connector (P_{tc}), and of $\dot{V}$ permit calculation of expiratory resistance (R_E) across the airway and endotracheal tube:[66,67]

$$R_E = \frac{(P_{alv} - P_{tc})}{\dot{V}_t}.$$

When alveolar pressure is referenced to P_{atm} instead of P_{tc}, R_E includes the external resistance across the expiratory valve as well.[25,68]

Like R_I, R_E is volume dependent, progressively increasing as the lung deflates. This volume dependency is especially evident in severely obstructed patients with dynamic airway collapse. Because expiratory airflow is multiphasic in such patients, several stop-flow measures of P_{alv} are needed to adequately characterize R_E over the entire tidal range.[60]

It is not commonly recognized that the resistance of the expiratory path can be calculated from P_{aw}

alone, assuming passive deflation.[60] Normally, pressure and volume decay exponentially during exhalation, at a rate determined by the deflation time constant. Therefore, assuming uni-exponential deflation characteristics, volume at any time (t) after exhalation begins (V_T) can be expressed as the quotient of the starting volume above equilibrium (ie, FRC) [$V_0 = \dot{V}_t + (AP \times C)$] and a power of e (the base of the natural logarithm system);[38,69,70]

$$V_t = \frac{V_0}{e^{kt}}.$$

In this expression, k (a constant) is the reciprocal of the time constant (τ), which itself is the product of R_E and Crs [$k = 1/\tau = 1/(R_E \times C)$]. Thus, if τ and C_{rs} are both known, R_E can be calculated: $R_E = \tau/C_{rs}$. Because volume and P_{alv} are approximately linearly related during exhalation, P_{alv} can also be expressed in the same exponential fashion:

$$P_t = P_0 e^{-kt}$$

where $P_0 = P_S$.[60]

Although τ can be determined from either the alveolar pressure × time or volume × time curves, determination for alveolar pressure should be preferred in the clinical setting because unlike volume, it inherently takes into account any component of alveolar pressure associated with hyperinflation (auto-PEEP). When pressure is used, unusually protracted exhalation times[38] are not required to release the volume trapped by hyperinflation before the "τ-based" R_E measurement is attempted. As an added advantage to using pressure rather than volume, P_{aw} can be measured (and P_{alv} estimated) in every patient, regardless of the ventilator's monitoring capability.

τ can be determined from P_S in conjunction with a post-occlusion P_{aw} recorded at any random time after exhalation begins:[60]

$$P_t = P_S \times e^{-t/\tau},$$

where (P_t) = recorded pressure at time t. Hence,

$$P_t/P_S = e^{-t/\tau}.$$
$$\ln (P_t/P_S) = -t/\tau.$$
$$\tau = t/\ln (P_S/P_t).$$
$$R_E = 1/[c \times \ln(P_S/P_t)].$$

This value for R_E is not a point measurement, but rather typifies resistance over the initial phase of deflation to the point of occlusion. In patients with dynamic airway collapse during tidal breathing, a one-compartment exponential model is a poor description of the expiratory decay of alveolar pressure because resistance in early exhalation differs considerably from that in late exhalation.[59] Imprecision can be reduced by modeling a two-compartment system using three occlusions.[60]

Endotracheal tube resistance. The pressure gradient across the ET tube can be estimated from published data or measured directly.[25,71] In-vitro studies of clean ET tubes of fixed length do not reflect such important patient-specific (in-vivo) problems as tube kinking, secretion buildup, or tube malpositioning. Thus, in-vivo resistance may be substantially greater than in-vitro data would suggest.[72] When accuracy is required, a small-bore catheter can be advanced beyond the carinal tip of the ET tube to sample tracheal pressure. When coupled with P_{aw} measured in the external airway, such tracheal pressures (P_T) enable calculation of trans-ET tube pressure ($P_{aw} - P_T$) and effective ET-tube resistance.

Impedance to Inflation

The overall difficulty in chest expansion, 'inflation impedance,' consists of resistive, elastic, and inertial components. Under conditions of passive inflation, P_{aw} sums all pressures needed to overcome these hindrances, and the integrated product of P_{aw} and $\dot{V}$ defines inflation impedance, or the work of chest inflation (W_I):

$$W_I = \int P_{aw}\, \dot{V} dt.$$

During passive inflation with *square-wave* (constant) flow, time and volume are direct linear correlates, so that the time axis can double as a volume axis, with T_I corresponding to V_T. Under these constant flow conditions, W_I is directly proportional to the area under the P_{aw} tracing measured over the time of inflation (t_I). Furthermore, mean inflation pressure (P_{avg}, the work/L) is simply W_B/V_T. With flow and V_T held constant, P_{avg} serves to index the inflation impedance of the respiratory system (Fig. 9). Average inspiratory pressure during passive inflation with square wave flow can also be estimated by *any* of the following:[1,73,74]

$$P_{avg} = P_D - \frac{(P_S - P_{alv\text{-}ee})}{2}.$$

$$P_{avg} = P_{aw} \text{ recorded at } t_i/2.$$

$$P_{avg} \cong 0.80 \times P_D.$$

$$P_{avg} = \left(\frac{V_T}{t_i}\right)R_I + \frac{V_T}{2 \times C_{rs}} + AP.$$

Under passive conditions, the P_{aw} tracing not only permits calculations of resistance, compliance, and external work, but can also provide valuable information that is lost when only needle manometer or digital measurements are recorded.[62] Upward inflection of the end-inspiratory segment of the P_{aw} tracing suggests alveolar overdistention and alerts the clinician to the risk of impending barotrauma if the unmodified ventilation pattern is continued.

Conversely, convexity in the *early* portion of the P_{aw} tracing suggests AP, volume-dependent resistance, or inflation hysteresis (due to alveolar collapse). Because each of these problems tend to resolve with higher end-expiratory volume, a high P_{aw} shoulder may predict benefit from judicious PEEP application (Fig. 10).

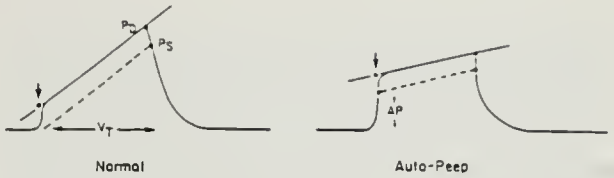

Fig. 10. Alteration in the airway pressure profile of a controlled constant-flow breath caused by auto-PEEP (AP). The "squared off" shoulder of pressure at inflation onset represents the sum of resistive pressure losses and the positive airway pressure necessary to counterbalance auto-PEEP. (Reproduced, with permission, from Reference 75.)

Work of Breathing

Measures of the external (mechanical) work of breathing do not account for isometric activity, poor mechanical advantage, or discoordinate contraction by opposing muscle groups. The contributions of these important components are better accounted for by measurements of the oxygen consumed by the ventilatory muscles or perhaps by integrated, rectified electromyography. At best, therefore, mechanical indices can only approximate the degree of respiratory muscle activity. However, offsetting this major disadvantage is the fact that external work is easily and precisely measured. Furthermore, if either mechanical work or the pressure-time product is related to its maximum achievable value, an excellent indication of ventilatory stress is obtained. Work, the product of inflating pressure and volume, can be determined by planimetry of pressure-volume plots or by electronic integration of the pressure-flow product.

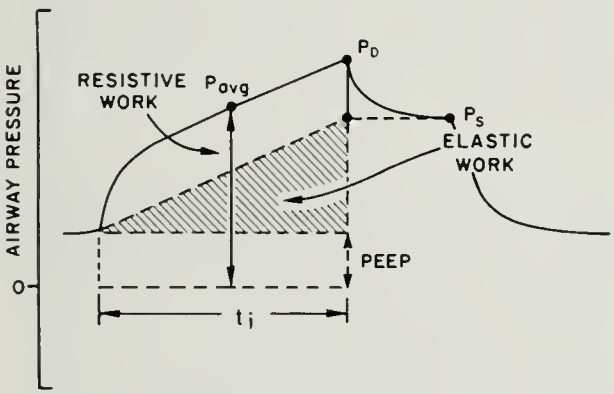

Fig. 9. During constant-flow mechanical ventilation under passive conditions, the average inspiratory pressure (P_{avg}), an index of inflation impedance, is comprised of elastic and resistive components. P_{avg} can be estimated from the airway pressure tracing (P_{aw}): $P_{avg} = P_D - (P_S - P_{alv\text{-}ee})/2$, where P_D = peak dynamic pressure, P_S = peak static (plateau) pressure, and $P_{alv\text{-}ee}$ = end-expiratory alveolar pressure. P_{avg} can also be estimated by multiplying P_D by a constant "k" ($k \cong 0.80$) or by measuring P_{aw} when only half of the inspiratory time ($t_i/2$) has elapsed. (Reproduced, with permission, from Reference 50.)

External mechanical work is performed across the airways, lungs, and chest wall in moving volume against frictional and elastic pressure gradients. Thus, under conditions of *passive* inflation, the work performed (by the ventilator) during a tidal inflation can be partitioned (Fig. 5):[76]

$$\text{resistive work} = \int_0^{t_i} (P_{aw} - P_{alv})\dot{V}dt \quad (1)$$
$$(airway)$$

$$\text{elastic work} = \int_0^{t_i} P_{alv}\dot{V}dt.$$

$$= \int_0^{t_i} (P_{alv} - P_{pl})\dot{V}dt + \int_0^{t_i} (P_{pl} - P_{atm})\dot{V}dt. \quad (2)$$
$$(lung) \qquad + \qquad (chest\ wall)$$

$$\text{total work} = \int_0^{t_i} (P_{aw} - P_{atm})\dot{V}dt. \quad (3)$$

During spontaneous breathing, the respiratory muscles must generate a total pressure gradient similar to that specified by this equation. Because the lung is an inherently passive structure, work performed across the airways and parenchyma can be quantified by the same expressions used above. However, the pressures generated by the respiratory muscles in expanding the chest wall are inaccessible to measurement, and therefore must be approximated.

Regardless of mode of ventilation (spontaneous or mechanical), P_{avg}, the sum of average resistive and elastic pressure components, can be estimated from a modification of the "equation of motion" of the respiratory system:[74]

$$P_{avg} = \left(\frac{V_T}{t_i}\right)R + \frac{V_T}{2(C_{rs})} + AP.$$

(Note that for the same $\dot{V}_E$, total work [$P_{avg} \times \dot{V}_E$] is diminished in proportion to the reduction in V_T, owing to a reduction in elastic work per liter.) P_{avg} is also the work per liter of ventilation:

When W_B = Work/breath

$$W_B = V_T + P_{avg}.$$

$$P_{avg} = \frac{W_B}{V_T}.$$

Spontaneous work of breathing. The direct measurement of the work of spontaneous breathing requires esophageal balloon placement for pleural pressure estimation, so that changes in the transpulmonary ('trans-lung') pressure (P_{lp}) can be determined during active efforts. The additional work of expanding the chest wall during a *spontaneous* (active) breath (W_{cw}) is usually approximated by the work performed to distend the passive chest by a similar volume.[77] This latter component can be quantified by direct planimetry of the *passive* $P_{alv\text{-}ee} \times$ volume plot, by electronic integration of the product of P_{es} (relative to the end-expiratory value) and inspiratory flow, or by calculating the product of V_T and the average pressure inflating the chest wall (P_{cw}):

$$P_{cw} = \frac{V_T}{2(C_{cw})} + AP\,\frac{(C_L)(C_{cw})}{(C_L + C_{cw})}.$$

At the bedside, the work of breathing during spontaneous ventilation can be estimated by three methods without complex equipment:

1. The equation of motion permits work estimation, if R_I and C_{rs} are measured.
2. Spontaneous work can also be estimated from $P_{aw} \times V_T$ planimetry (or P_{aw}-t_I planimetry during constant flow) of passive, mechanically delivered breaths matched for mean flow and V_T with their spontaneous counterparts.[78]
3. Finally, spontaneous work can be roughly estimated as a simple fraction (k = 0.80) of peak dynamic pressure (P_D) of a controlled mechanical breath (square-wave flow) if average $\dot{V}_I$ and V_T are matched to their spontaneous counterparts. If breaths are not matched for flow and volume, P_D must be scaled for these differences.[79]

Work during machine-assisted ventilation. Given that the mechanical ventilator is fully capable of breathing for the patient, it might be reasoned that the patient's work during machine-assisted ventilation should be negligible, apart from the minor effort involved in triggering machine support. This may well be true when the patient is very comfortable and unstressed. In practice, however, muscle contraction continues throughout most of inspiration when the patient is dyspneic, with depth and duration that depend on muscle strength and ventilatory drive.[10] During constant-flow ventilation, evidence of spontaneous respiratory activity is apparent in the P_{aw} tracing, either in distortion of its normal trapezoidal shape or in the variance of its peak cycling pressure.[1,62]

Respiratory muscle activity during triggered machine cycles can be influenced by trigger sensitivity, V_T, $\dot{V}$, and $P_{alv\text{-}ee}$, to the extent that each influences patient comfort and dyspnea.[10,80] Surprisingly, perhaps, the extent of work performed by the patient during a ventilator-assisted cycle does not depend in any important way on the resistance or compliance of the chest—provided that machine-delivered flows and tidal volumes meet or exceed patient needs.[10] When patient demand outstrips machine flow (and volume), the patient not only works against his own intrinsic inflation impedance but also against the resistance offered by inspiratory valves and circuitry.[81] Such events are noted by depression of the airway pressure tracing below the end-expiratory pressure baseline.

Effect of auto-PEEP on work. Auto-PEEP (AP) often develops in the setting of airflow obstruction,

high minute ventilation requirement, and dynamic airway collapse. Very slow expiratory flow continues at end-exhalation, driven by positive alveolar pressure. To initiate inspiratory flow, positive (expiratory) pressure must first be counterbalanced; only then can pressure be generated in the central airway that is less than the baseline end-expiratory pressure set on the machine. This counterbalancing pressure is provided by the inspiratory muscles, so that AP presents a threshold load to each inspiratory cycle, whether spontaneous or machine assisted.[59] In a sense, AP blunts *effective* triggering sensitivity.

During *spontaneous* cycles, AP adds a threshold level of muscle tension to that required to ventilate, increasing workload. During triggered, machine-assisted breaths, AP reduces the effective sensitivity of the demand valve to a value that approximates the sum of AP and the set trigger sensitivity. Because trigger sensitivity is a prime correlate of drive and work during machine-assisted breathing,[80] AP increases the W_B during these cycles as well. Because many patients in whom AP develops are flow-limited during tidal breathing, pressure added downstream to the point of collapse (eg, in the central airways) may improve machine sensitivity without notably increasing P_{alv}. Respiration improves effective trigger sensitivity by bringing P_{aw} closer to P_{alv}. Thus, the impact of AP on W_B can often be attenuated without markedly changing P_D or P_S, simply by applying low levels of PEEP (always less than the original level of AP).[59,82,83]

Techniques for measuring patient work during machine-aided breathing cycles—*Volume-cycled breathing.* The amount of external work performed by the patient during machine-assisted inspiration (W_p) can be quantified from plots of V_T against either P_{aw} or P_{es}[10,80] (Fig. 11). (Because all muscular effort influences externally measured work by first changing P_{pl}, either P_{aw} or P_{es} can be used as the reference pressure in the work calculation.) Assuming that equal amounts of external work are required during controlled and patient-triggered breaths of the same flow profile and volume, W_p is quantified by the difference between the pressure-volume plots generated in these modes. Thus, W_p can be viewed as the decrement in machine work that occurs during a triggered cycle in accomplishing an identical inflation pattern.[10,80] Using the planimetric method:

Spontaneous work = [passive work]
[1 – area under triggered curve/area under passive curve].

Accurate estimation of W_p requires the passive curve (to which the assisted cycle is compared) to have similar impedance characteristics for the respiratory system.

Techniques for measuring patient work during machine-aided breathing cycles—*Pressure support ventilation.* During pressure support (unlike assisted mechanical ventilation), airway pressure rather than flow is held constant, and the depth and duration of inspiration are under patient control. Despite these differences, work can be computed from P_{es}-volume plots in a fashion similar to that described for assisted mechanical ventilation. External work

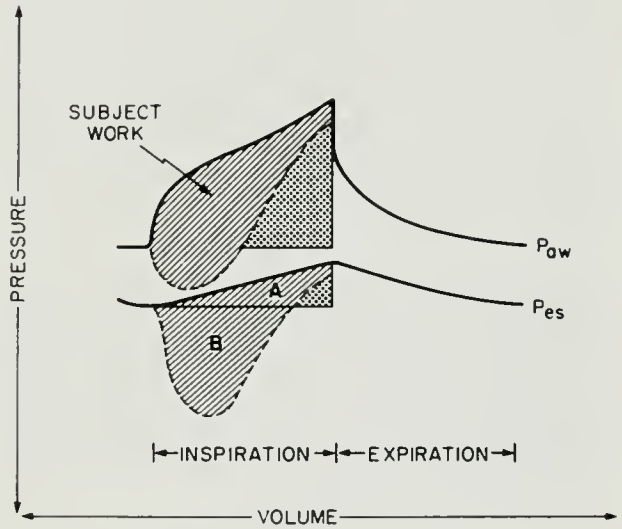

Fig. 11. Patient work (W_p) during triggered breathing can be quantified by subtracting the planimetered area under the airway pressure tracing (P_{aw}) of a triggered breath from that of a controlled mechanical breath, provided equal flow and volume are delivered. Alternatively, W_p can be quantified from differences in planimetered areas between triggered and controlled esophageal pressure-volume plots, provided equal flows and volumes are delivered. (——) represents a controlled breath and (----) represents an assisted triggered breath. Area A represents work performed by the patient in expanding the chest wall. Area B represents the work performed by the patient in expanding the lungs and moving volume against resistance. When P_{aw} is used to compute W_p, triggered and controlled breaths must have equivalent inspiratory resistance. (Reproduced, with permission, from Reference 62.)

performed across the lung and inspiratory circuit is $\int P_{es}\dot{V}dt$, or the planimetered P_{es} × volume area. To compute the total W_p during pressure support ventilation (W_{psv}), a component for chest-wall expansion must be added. Because the passive pressure-volume relationship of the chest wall is essentially linear, whatever its rate of expansion, the chest-wall component (W_{cw}) can be computed as:

$$W_{cw} = \frac{(V_T)^2}{2C_{cw}}.$$

Pressure-Time Product

Volume must be moved in order to accomplish external work; measures of external work do not account for isometric components of muscle contraction. Thus, a patient with severe airflow obstruction accomplishes very modest external work, despite great exertion. As a more extreme example, attempts to inspire against an occluded airway, however vigorous, develop muscle tension but accomplish no measurable external work at all. For these reasons, the product of developed pressure and duration of muscle contraction (the "pressure-time product") often reflects muscular effort more faithfully than it reflects W_p.[84-86]

Pressure-time product is the product of average inflation pressure and duration of contraction, as measured by planimetry of the P_{es} curve. As with the work measurement, an adjustment for chest wall expansion is desirable. In the determination of pressure-time product, planimetry begins from the point of effort onset, not zero flow. This is important in the presence of auto-PEEP.

Ventilatory Demand

The energy demand of the respiratory system is the product of energy demand per cycle (as determined by the inflation impedance, W_I, already discussed) and breathing frequency. Expressed another way:

Total energy demand = energy demand/L of ventilation × $\dot{V}_E$.

Minute ventilation varies with carbon dioxide production, dead-space fraction, and ventilatory drive. The importance of $\dot{V}_E$ to the work equation is crucial to keep in mind, as the ventilation requirement can often be manipulated more effectively than inflation impedance.

Ventilatory Capability

Drive To Breathe

Utility of measurement. In the clinical setting, ventilatory drive is a potentially valuable but seldom utilized measurement. Drive measures correlate with dyspnea, patient work, $\dot{V}_E$, and ability to wean from mechanical support.

Indices of drive—*Minute ventilation, average inspiratory flow.* In subjects with normal strength and normal chest mechanics, $\dot{V}_E$ and mean inspiratory flow (V_T/t_i) correlate well with the output of the respiratory neural center, both for the same subject and in cross-subject comparisons.[87] Critically ill patients, unfortunately, often have abnormal respiratory mechanics, decreased strength, or reduced endurance. With ventilation unsupported, many cannot maintain $\dot{V}$ or V_T in proportion to drive. In these patients, the strong correlations between drive and $\dot{V}_E$ and V_T/t_i that apply to normal subjects break down.[47]

Indices of drive—*Mouth occlusion pressure at 100 ms ($P_{0.1}$).* The $P_{0.1}$ correlates well with the respiratory center's neural output, both for normal subjects and for many types of patients.[8,88] As a static (isometric) measurement, $P_{0.1}$ is not influenced by respiratory mechanics (impedance). However, the $P_{0.1}$ measurement is affected by muscle strength and hence by the lung volume at which occlusion occurs.[9,89] Therefore, in such patients, the $P_{0.1}$ may not accurately reflect the drive to breathe unless it is related to the maximal rate of pressure development ($P_{0.1max}$). Nonetheless, the $P_{0.1}$ is a relatively simple index to measure and appears to hold promise in the evaluation of the weaning patient.

As already noted, the majority of ventilators that use demand valves impose a measurable delay before the demand valve opens, especially when trigger sensitivity is reduced. Pressures generated 100 ms into this pre-inspiratory period of isometric contraction correlate with formally measured $P_{0.1}$, theoretically, providing a measurement that can be made without the need for circuit modification or disruption of the breathing rhythm.[11] Thus, although such pressures must be recorded rapidly and with great precision, the $P_{0.1}$ is potentially measurable in the majority of ventilated patients.

Strength

Measuring muscular strength—*Maximal inspiratory pressure.* The maximal inspiratory pressure (MIP) maneuver can be used to gauge the strength of the respiratory muscles. As a static (isometric) measurement, however, the MIP exceeds dynamic capability. The disparity between maximal static and maximal dynamic pressures widens as $\dot{V}_E$ and flow velocity increase (Fig. 12). Furthermore, full

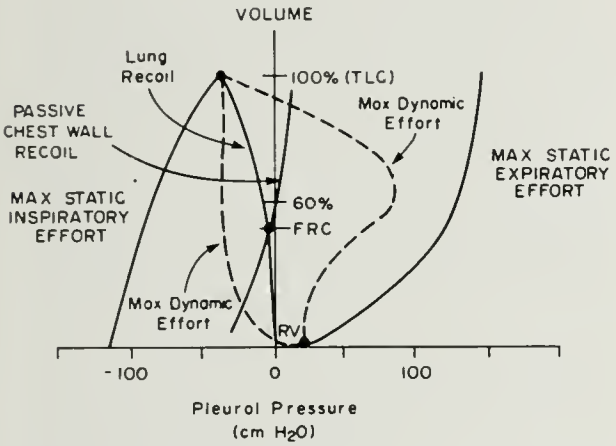

Fig. 12. Dynamic and static pleural pressure-volume relationships. Data from Figure 2 are replotted with a reduced pressure scale. Also shown are esophageal pressures generated by maximal expiratory and inspiratory efforts with the airway occluded and the pressures during maximal dynamic efforts begun from the extremes of volume with the airway open. Area between the dynamic curves defines a maximal performance envelope for the respiratory muscles. (Reproduced, with permission, from Reference 90.)

cooperation is necessary to elicit maximal effort, and the pressure that develops bears a strong negative correlation to increases in lung volumes above FRC.[5] Recently, our group has demonstrated that an MIP measured with a one-way valve after CO_2 stimulation closely approximates the maximal voluntary MIP, provided that CO_2 stimulation successfully elicits a high level of drive.[91]

Measuring muscular strength—*Vital capacity.* Vital capacity also provides information about muscle strength, but this measure, too, is profoundly influenced by lung mechanics and effort. Furthermore, when the patient is fully cooperative and chest mechanics are normal, muscle strength must be reduced to 20% of the normal value before significant reductions in vital capacity occur on a strength basis alone.[92] As noted earlier, a "stacked vital capacity" can be measured in poorly cooperative patients using a one-way valve, applied in sequence to the inspiratory and expiratory limbs of the breathing circuit.[49] Similar peak pleural pressures must be generated to achieve equal volumes by either stacked or conventional methods. The difference is that "stacked" volumes but not inspiratory pressures are sustained. Like the standard vital capacity maneuver, the stacked vital capacity remains sensitive to alterations of chest mechanics.

Endurance

Utility of measurement. When one is concerned about a patient's ability to sustain ventilation without assistance, energy reserve (endurance)—not strength—is the primary issue. Although strength measurements tend to correlate well with endurance, the predictive relationship is not exact (largely because the true load placed on the muscles varies with $\dot{V}_E$ and mechanics). As a general principle to assess endurance, strength measures and drive measures must be compared to their maximal values.

Measures of endurance—*Minute ventilation to maximum minute ventilation ratio.* The energy reserve of a cooperative subject can be assessed by the ratio of $\dot{V}_E$ to maximum voluntary minute ventilation ($\dot{V}_E$:MVV).[93] Unfortunately, however, full patient cooperation, a rare attribute of the critically ill patient, is required for the MVV maneuver. Therefore, this index is helpful only when the ratio is deemed depressed. Ratios less than 1:2 reliably indicate that spontaneous ventilation can be sustained indefinitely after machine withdrawal provided that $\dot{V}_E$, strength, and mechanics do not change adversely over time.[94]

Measures of endurance—*Electromyographic power spectrum.* Research data suggest that endurance can be assessed by the power spectrum or frequency analysis of the electromyographic (EMG) signal.[95,96] A decrease in the ratio of high-to-low-frequency content suggests that the respiratory load is a relatively large and perhaps overwhelming burden. The ratio of the integrated EMG signal to average inflation pressure (P_{avg}) also increases when

subjects can no longer endure the respiratory stress; a worsening disparity between integrated EMG (an indicator of stimulation intensity) and the resultant inspiratory pressure suggests fatigue.

Measures of endurance—*Mouth occlusion pressure ($P_{0.1}$).* The level of drive may reflect energy reserves for spontaneous breathing. In a small study of patients with chronic obstructive airways disease abruptly withdrawn from mechanical support, Sassoon et al[97] found that a $P_{0.1} > 6$ cm H_2O predicted that ventilatory reserves were inadequate to meet the challenge of unsupported ventilation. The response of $P_{0.1}$ to CO_2 stimulation may correlate better with endurance. The results of another study by Montgomery et al[98] implied that ICU patients who fail to demonstrate a notable increase in $P_{0.1}$ when stimulated by carbon dioxide fail to wean successfully. Although the implications of both of these intriguing studies are limited by sample size, they do suggest a role of drive indexes in the evaluation of the weaning patient. Recent European work suggests that $P_{0.1}/P_{0.1max}$ is an excellent predictive index for weanability (personal communication from R Fernandez and S Benito, Barcelona, Spain, November 1989).

Measures of endurance—*Breathing pattern.* As fatigue develops, tachypnea and discoordinated breathing become prominent features of the breathing rhythm.[48,99] This topic is addressed at length in a companion paper[100] and will not be discussed further here.

Measures of endurance—*Pressure-time index.* The pressure-time index (PTI), a correlate of O_2 consumption,[101] is defined as:

$$PTI = (\bar{P}_{di}/P_{max})(t_i/t_{tot}),$$

where $\bar{P}_{di}$ is the mean transdiaphragmatic pressure developed during a tidal breath and t_i/t_{tot} is the inspiratory time fraction. In experimental studies of normal subjects, the PTI exhibits a strongly inverse correlation with diaphragmatic endurance; inability to sustain a targeted pressure for longer than 45 minutes was noted when PTI values exceeded 0.15 to 0.18.[102,103] Although the fatigue zone for PTI has only been defined for the diaphragm, there is reason to believe that a similar, if not identical, principle that a similar, if not identical, principle might apply to the entire respiratory system if either esophageal pressure or calculated average transrespiratory pressure (P_{avg}) is used in place of mean transdiaphragmatic pressure ($\bar{P}_{di}$).[104] If so, the PTI might serve as a valuable clinical guide to the timing of ventilation. A $\bar{P}_{di}/P_{max}$ ratio of 0.4 (or a $P_{es}/P_{es\text{-}max}$ ratio of −0.6) cannot be indefinitely sustained. Because t_i/t_{tot} seldom deviates markedly from 0.4, the P_{avg}/P_{max} ratio contains most of the useful information contained in the pressure-time index.

Milic-Emili has emphasized that weaning outcome may be predictable from the PTI values estimated from V_T, t_i/t_{tot}, $\dot{V}_I$, and MIP.[73] As illustrated in the data from Tobin et al,[48] successfully weaned patients tend to have pressure-time index values < 0.15. All components of the PTI can be assessed with varying degrees of accuracy at the bedside of a cooperative subject. As discussed earlier, P_{max}, or MIP, may be estimated using CO_2 stimulation and a one-way valve system. Duty cycle (t_i/t_{tot}) can be easily recorded from the P_{aw} tracing. The average inspiratory pressure (P_{avg}) of a spontaneous breath can be estimated using any of the four methods listed earlier. Despite the relative ease with which the pressure-time index can be computed, its accuracy and value in predicting the outcome of a weaning trial remains unconfirmed at the present time.

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Marini Discussion

Krieger: If you have the esophageal balloon in place, why not just use the esophageal balloon to measure auto-PEEP?

Marini: If the objective is to quantify end-expiratory *alveolar* pressure, I think you have to look at the *difference* in esophageal pressure before you know what it is. I can put an esophageal balloon in a patient with COPD and measure a resting value of +5.

Krieger: Correct, and isn't that auto-PEEP? Because it's positive alveolar pressure at end exhalation . . .

Marini: Bruce (Krieger), I don't think the absolute esophageal pressure and auto-PEEP are the same thing—because the inside pressure of the lung and the outside pressure of the lung is the difference that we're talking about here during spontaneous breathing. You could have +5 inside, and –5 outside at end exhalation—and –5 (the esophageal pressure) is not the auto-PEEP, right? It's the esophageal pressure *change* needed to counterbalance that positive alveolar pressure that's important.

Krieger: If you have zero at the central airways and you have +5 in the esophageal balloon, you're saying that +5 is not a reflection of auto-PEEP?

Marini: Sometimes it is, and sometimes it isn't. Auto-PEEP is the sum of pleural and lung recoil pressures at end-exhalation. One of the things about the esophageal balloon is that the absolute values can sometimes be absolutely unrelated to the global average pressure that surrounds the chest, especially in a recumbent position. However, as a reflection of differences in that average pressure ($\bar{P}$), it's an acceptable measurement.

Kacmarek: I agree 100%; you can measure $\bar{P}$ in someone whose chest wall you're passively inflating. With spontaneously breathing patients, it's not always easy to take over and passively inflate the chest wall. At the bedside, how do you come up with $\bar{P}$ in someone breathing spontaneously?

Marini: In someone who is breathing spontaneously, what you need to do is figure out what all the components are of the modified equation of motion. I assume that you have reliable numbers for resistance and compliance. You can easily measure tidal volume; you can also easily measure inspiratory time. Let's take an example: the visual expression for average inspiratory flowrates, V_T/t_i. Another expression for average inspiratory flow is minute ventilation ($\dot{V}_E$) over t_i/t_{tot}. Okay? Breathing 12 L/min and t_i/t_{tot} 0.5, then this average inflation flowrate is going to be 24 L/min. And if you divide that by 60 to get what it would be per second and multiply it by the resistance value that you have calculated, then you know the frictional component. Similarly, if you know their average tidal volume and compliance, you can compute the tidal elastic component. To finish the computation, you need an estimate of auto-PEEP.

Kacmarek: So, the critical variable—you're saying—is simply being able to get to the point where you can get at least one good breath where you can 'plateau it,' get a passive inflation, and hopefully come up with (in addition to that) an evaluation of the amount of auto-PEEP by an expiratory occlusion or whatever method you're using noninvasively?

Marini: Right. And I'm not so sure that our spontaneously breathing patients have much auto-PEEP (at least not at the minute ventilations at which they're likely to wean).

Kacmarek: Right, just a couple of centimeters.

Marini: A few centimeters.

Krieger: John (Marini), what are your thoughts about Milic-Emili's inspiratory effort quotient (IEQ), which was derived from the same theoretical basis?[1]

1. Milic-Emili J. Is weaning an art or a science? (editorial). Am Rev Respir Dis 1986;134:1107-1108.

Marini: Milic-Emili's IEQ is simply a modified tension-time index. Multiply the peak dynamic pressure of the ventilator by 0.75, and you have an estimate for average inflation pressure, the $\bar{P}$ we've been talking about. Manipulate that around a little bit, factor in the t_i/t_{tot}, and you get the IEQ. I think it's basically correct. I think I should point out, however, in principle, that although it's correct you must use the variables that apply to the spontaneously breathing patient. That's generally true whenever you're thinking of work of breathing or pressure-time product or anything like that. By the way—I think that pressure-time product is a better thing to look at than is work of breathing. If you're relating work of breathing to the absolute amount of work that someone can do, that's fine; but, if you're interested in how much oxygen consumption ($\dot{V}_{O_2}$) is occurring with respiratory muscles, I think the pressure-time product parallels $\dot{V}_{O_2}$ much more closely than does the work of breathing, especially if the afterload is changing. This average pressure is used in both calculations.

MacIntyre: John (Marini), I want to thank you for these ideas because I think they're very clever. We looked at our long-term weaning patients and found that using your technique, a $\bar{P}/P_{max}$, or P_{avg}/P_{max}, of 45% was a very useful predictor of when people are ready to be weaned.[1]

Another point about the technical aspects of measuring auto-PEEP—these calculations utilize simultaneous measurements of flow and pressure. It's crucial that the response characteristics of the flowmeter and the pressure transducer be the same. Otherwise, a measurement of pressure done at one place in the circuit with a transducer that has a response time unlike that of a flowmeter someplace else will generate different lag times and phase delays that may lead to substantial errors. The net result would be to make you think you have more auto-PEEP than you really do. In fact, a measured delay between pressure drop and inspiratory flow may, in addition to auto-PEEP, also reflect demand-valve functioning or artifact due to different sensor sites and characteristics.

1. Li-Ing H, MacIntyre N. Physiologic determinants of ventilator dependence. Chest 1989;96(2, Suppl):300S.

Kacmarek: I don't know of any ventilator that is capable of measuring or giving you the data you need in a spontaneously breathing patient.

MacIntyre: That's right.

Kacmarek: But, if they're breathing spontaneously, you have to add instrumentation at the airway to do this.

MacIntyre: I've observed people trying to take your ideas, John (Marini), and use the ventilator flow signal to imply that auto-PEEP is present. I think your concept of auto-PEEP is correct, but I think trying to measure it with the tools we currently have is extremely difficult if not

impossible. And I'm afraid it can be misleading.

Kacmarek: In a spontaneously breathing patient, absolutely.

Marini: Now I'm confused. How do you measure auto-PEEP in a spontaneously breathing patient? With an esophageal balloon and a pneumotachometer?

Kacmarek: What Neil (MacIntyre) is describing is the practice of taking the analog signal from the ventilator and using that as the flow signal and comparing that to the esophageal pressure change. Because of delays, I don't think you can do that. I think you have too much of the phase delay. There is no way that you'd be able to come up with an appropriate auto-PEEP measurement.

MacIntyre: I agree.

Kacmarek: You need to put a pneumotach at the airway.

MacIntyre: I agree.

Marini: Chuck (Burke), do you agree? When we use airway pressure and flow signals, is there a great deal of phase difference between the pressure measurement and the flow measurements?

MacIntyre: You're talking about small periods of time of no flow, and I am afraid that period of no flow could very easily be . . .

Burke: We do everything at the airway.

MacIntyre: Well, that's my point. If you do everything at the airway, you're okay.

Marini: If you monitor pressure at the airway opening, which everybody can do . . .

Burke: All you need to do is to attach the pressure transducer to the temperature port of the ventilator circuit.

MacIntyre: That's the pressure. I'm talking about flow.

Marini: Neil (MacIntyre), where do you measure flow?

MacIntyre: Well, most of the ventilators that I'm aware of have their flow measured somewhere in the guts of the machine—closer to the demand valve than to the patient.

Marini: Okay. Well, you know, if you're relying on the *inspiratory* flow signals generated in the machine, I accept your point. If you're talking about flow as we have been doing it, we always do it centrally, at the same place where we measure pressure.

MacIntyre: John (Marini), I'm saying you're physiologically correct.

Marini: And the delays are very small.

MacIntyre: No, physiologically you're correct. I'm just trying to clarify the fact that this is not something you can just grab off the ventilator flow signal.

Marini: I accept that. You have to pay close attention to the potential for phase delay.

Chatburn: I want to get to the point where you were describing the two different kinds of values you can get for compliance. If I understand what you're saying, you're really talking about the difference between a dynamic compliance and a static compliance.

Marini: Right.

Chatburn: I'll ask you the question that you asked me during the discussion following my presentation: How much information can you get out of comparing those two values and how useful is that sort of thing?

Marini: Potentially there may be some information there. The difference between such dynamic and peak static pressures is usually 15% or less of the total deflection at the flows we usually use. So, I think we're looking at a fairly small pressure drop here. I do think that potentially there is some information to be gained, however, because of distribution of volume questions. These may often be as important as what the absolute volume is. How is volume being distributed and how much recruitment is going on? If my tidal volume is normal but it's all going to one lung, that's not good. And if I increase peak pressure, then all I do is hyperexpand an already distended lung, and I don't want to do that. So, I think it might be useful to pay attention to these different types of compliances as well as to the absolute volume delivered.

Chatburn: As far as the mechanics of obtaining the numbers, I think you're not limited to looking at those very small pressure changes or very small time changes—I think you can get the same information by the two different techniques of determining the static compliance as you have shown here—that is, by determining static mechanics using an inspiratory-hold maneuver and by obtaining a dynamic number by fitting a linear regression equation to the whole curve. I think you'll come up with the same discrepancy, rather than trying to pick it off with that kind of a flow-pressure signal.

Marini: How would you do that again, Rob (Chatburn)? You would just use different tidal volumes?

Chatburn: No, you could use the same volume; the typical example here is the way that the Puritan-Bennett 7200 gives you a dynamic compliance vs a static compliance. The one uses the plateau and inspiratory-hold maneuver; the other uses a linear regression equation to which you fit a bunch of datapoints over the inspiration.

Burke: This is just a comment: I know that Dean (Hess) has worked with a test lung like we use in our laboratory. We have set up a two-compartment lung, each having two very different time constants—very different—we were surprised that, although these lungs were not reaching a plateau by any means, the digital readout on all of the ventilators told us that we had a plateau.

Hess: Yes.

Burke: It's just something to be aware of. Looking at how that plateau responds as you leave it on can give you some idea of heterogeneity of the lung. We were surprised to see a readout. We had clinicians who would write these numbers down on the

ventilator check sheets, but there was no real plateau.

Marini: I think that emphasizes that the airway pressure tracing can be extremely helpful.

Branson: I think that's the whole key here. Nobody is arguing with what you're measuring, how you're measuring it, or how you report it—the point is, though, 97% of respiratory care practitioners do not have a dedicated system of pneumotachograph and differential pressure transducers, recorders, and computer systems, to collect this data. They're going to read it off the ventilator; and I think the thing we need to find out here and maybe work on in the future is, Can ventilators give this information? If it's not reliable, is it at least consistent? And to be consistent, what does it have to be? Rob (Chatburn) has done all of this work [in late 1989]; so he may want to comment. The Hamilton will give you compliance measurements if you have the plateau and if you use the square waveform, regardless of whether it's a patient-initiated or totally passive breath. The IRISA will only give it to you in control—assuming that that breath was not spontaneously initiated—which I assume would be the best way to do it. And I think the other ventilators are each doing it in a different fashion; some will even give you a compliance from pressure-supported breaths. I think that may be the biggest thing we can help with here—not the measurement and how useful it is, but how can we make it clinically applicable for the average everyday therapist.

Chatburn: All of the ventilators have different rejection criteria for when they will and will not analyze the data. And some, like those of the Bear 5, are tight rejection criteria; and, some like the 940—there's still an add-on package for that Siemens—essentially have no rejection criteria. You always get numbers, regardless of what's happening.

Kacmarek: I don't think there are any data, though. The only data that I've found that compare results with manual techniques to those using the ventilator were published and presented as an abstract at the ACCP meeting that I mentioned.[1] Correlation was extremely good—0.97 and up between the two techniques—although there was no agreement data. It's hard to say what that meant, but that's the only piece of information that I'm aware of in the literature—that one little abstract. I personally don't trust the ventilator systems and would prefer to do it manually—just like Rich (Branson) has proved that the inspiratory force measurement on the Puritan-Bennett 7200a is a problem.[2] You know the systems are designed and tested in the laboratory, but I'm not sure that their use is appropriate in every patient or that we should accept that data across the board.

1. Korst R, Orlando R, Yeston N, Molin M, DeGraff A, Gluck E. Validation of respiratory mechanics software microprocessor controlled ventilators (abstract). Chest 1989;96(2, Suppl): 255S.
2. Branson RD, Hurst JM, Davis K, Campbell R. Measurement of maximal inspiratory pressure: A comparison of three methods. Respir Care 1989;34:789-794.

Marini: I agree with that. I think we have two missions: One is to improve the science and the other is to improve its application.

Kacmarek: Exactly.

Marini: And it's incumbent upon us to criticize what's out there as well as to elucidate the techniques.

Ventilatory Pattern Monitoring: Instrumentation and Applications

Bruce P Krieger MD

Introduction

Computers introduced a multitude of technical innovations into the field of medicine, perhaps most noticeably in the cardiac intensive care unit (ICU) where continuous and complex electrocardiogram (ECG) equipment is present in the smallest ICU and where even cardiac balloon pumps are commonplace. Until a few years ago, respiratory monitoring remained relatively primitive and intermittent, consisting of arterial blood gas studies, bedside spirometry, chest radiographs, and periodic visual observation.[1] In my opinion, this situation is analogous to monitoring a patient with a life-threatening cardiac arrhythmia by taking his pulse every 2 hours and performing an ECG three times a day! Fortunately, the monitoring of pulmonary patients is becoming more sophisticated and easier, as this conference attests.

Ventilatory pattern monitoring, or breathing pattern analysis, is actually the oldest and the newest means of monitoring the pulmonary patient. Visual observation, or 'eyeballing the patient' to estimate the patient's work of breathing, is the cornerstone of modern microprocessor-based breathing pattern monitors such as the respiratory inductive plethysmograph. I will briefly review both the technical and the clinical aspects of the breathing pattern monitors that are currently available. For additional information, I refer the reader to recently published chapters on noninvasive respiratory monitoring that Sackner and I have written.[1,2]

Abbreviations Used in this Paper

AB	— Abdomen
ECG	— Electrocardiogram
FRC	— Functional residual capacity
FREQ	— Respiratory frequency
MVS	— Mechanical ventilator support
NIMU	— Noninvasive monitoring unit
NIP	— Neck inductive plethysmograph
$PEEP_I$	— Intrinsic positive end-expiratory pressure, or auto-PEEP
$\%RC/V_T$	— % that rib cage contributes to tidal volume
RC	— Rib cage
RIP	— Respiratory inductive plethysmograph
TCD/V_T	— Total compartmental displacement/tidal volume
TGV	— Thoracic gas volume
V_T	— Tidal volume

Instrumentation

A subject's ventilatory pattern may be monitored by: (1) visual observation, (2) volumetric measure-

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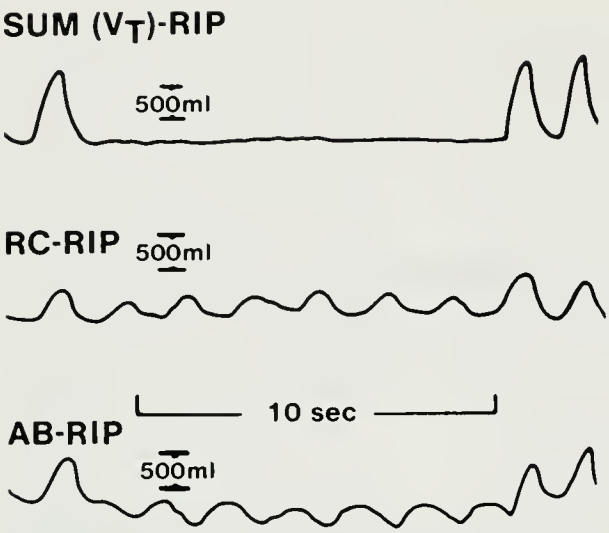

Fig. 1. Respiratory inductive plethysmography tracing of an isovolume maneuver. With the glottis closed, the RC and AB compartments move in opposite direction, resulting in a flat (apneic) sum (V_T) signal. (Reprinted, with permission, from Reference 1.)

ments made by a physical connection to the patient's mouth or airway (spirometers, pneumotachograph systems), (3) transducers placed about the face to detect airflow; and (4) transducers placed around or on the torso (bellows pneumographs, differential linear transformers, impedance pneumographs, inductance circumferential transducers, magnetometers, mercury-in-silastic strain gauges, and respiratory inductive plethysmographs). I will not discuss visual observation, but refer you to the companion paper by MacIntyre.[3] Also, because spirometers (although accurate) are unsuitable for continuous use, I will not discuss their application. Except for brief periods of application useful for research purposes, pneumotachographs can be used continuously only with intubated subjects. Although thermistors and thermocouples placed at the nose or mouth are used for continuous monitoring, their utility in breathing pattern analysis is limited because they produce only qualitative signals (and therefore are unable to accurately distinguish between periods of apnea and periods of hypopnea) and because they cannot distinguish a central from an obstructive event. Therefore, I will concentrate on those devices that can be placed on or around the torso for monitoring ventilation.

Konno-Mead Principle and Calibration Procedure

More than two decades ago, Konno and Mead[4] demonstrated that the open respiratory system behaved approximately as a simple physical system consisting of two independently moving parts—the rib cage (RC) and the abdomen (AB). During normal respiration, the diaphragm descends and displaces the abdominal contents while the RC expands. By appropriately calibrating the gains of the RC and AB signals from a magnetometer, Konno and Mead and later Sharp et al[5] were able to approximate tidal volume (V_T) in normal subjects by summing RC and AB movements. All respiratory monitoring devices that are placed around the torso are calibrated using the Konno-Mead principle or a variation thereof. If appropriately set, the sum of RC and AB movements will equal zero during an isovolume maneuver[4] that is accomplished by gently shifting volume to and from the RC and AB compartments while the airway is occluded (Fig. 1). Because the isovolume maneuver

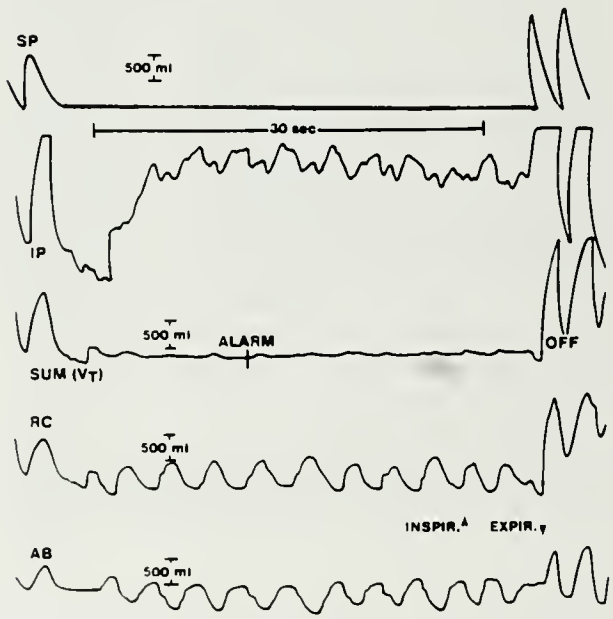

Fig. 2. During a simulated obstructive apnea (as evidenced by respiratory inductive plethysmography [RIP] signals showing rib-cage [RC] and abdominal [AB] movements in opposite directions, resulting in a flat sum [V_T] signal and no volume measured by simultaneous spirometry [SP]), the signal from an impedance pneumograph (IP) continues to show motion because of RC movement. Because RIP is calibrated based on two degrees of freedom, it appropriately senses an apnea and alarms, whereas the IP fails to alarm because it falsely interprets RC movement as a breath.

requires either a trained subject or an intubated patient in whom the endotracheal tube can be occluded during quiet breathing, alternative calibration procedures have been developed.[6–9] All calibration methods have been vigorously assessed only with respiratory inductive plethysmography.[2] The easiest and most accurate method to use clinically is the natural breathing, single-posture procedure based on isovolume principles. This method remains accurate and yields results within 20% of spirometric V_T despite changes in posture. In addition, it does not require spirometric validation for clinical decision making or trained subjects.[2,9]

Transducers Placed on or around the Torso

The impedance pneumograph, although widely used, is the only torso device that cannot utilize the Konno-Mead principle because it relies on passing a low-amplitude, high-frequency, alternating current between two electrodes placed on the chest only. Therefore, as reported in an abstract from our group,[10] it can neither reliably distinguish the chest motion that occurs during an obstructive event from an actual breath (Fig. 2) nor be relied upon to distinguish a strong ECG signal from an actual breath in many situations[11] or after a patient's position has been

Table 1. Components, Characteristics of Components, and Overall Assessment of Respiratory Monitoring Devices Placed around the Torso

Component and/or Characteristics	Bellows Pneumograph	Differential Linear Transducers	Inductive Circumferential Transducers	Magnetometers	Mercury-in-Silastic Strain Gauges	Respiratory Inductive Plethysmographs*
Transducer						
Comfortable	±	+	+	+	+	+
Stable and durable placement	±	+	+	+	−	+
Device Malfunction Alarm	−	−	−	−	−	+
Rate Detector						
Digital indicator	−	NA	NA	−	−	+
alarm	−	NA	+	−	−	+
Apnea Detector						
Irrespective of body posture	−	−	−	−	−	+
Obstructive vs Central	±	±	±	±	−	±
False alarms uncommon	NA	NA	±	NA	NA	+
Hypopnea Detector	−	−	−	±	NA	+
Usefulness & Accuracy						
Intensive care	−	−	−	−	−	+
Intermediate care	−	−	−	−	−	+
Sleep Laboratory	+	+	±	+	+	+

+ = present; − = absent; NA = not available.

*Using natural breathing calibration and microprocessor available on Respigraph (NIMS Inc, Miami Beach FL).

altered.[10] Because of these drawbacks, a recent panel on infantile apnea monitoring[11] seriously questioned its utility in its present form for detecting obstructive events in infants at high risk for sudden death (SIDS).

Noninvasive devices placed around the torso to monitor respiratory movements include bellows pneumographs, differential linear transformers, inductance circumferential transducers, magnetometers, mercury-in-silastic strain gauges, and respiratory inductive plethysmographs (RIPs). RIPs and magnetometers have had the widest research applicability, although all the listed devices are used in sleep disorders centers. Their features are compared in Table 1. Because the RIP is the only device that has been used extensively for long-term, continuous monitoring in the clinical setting, I will describe it in further detail.

The RIP utilizes two elastic cloth bands into which insulated Teflon wire is sewn in a sinusoidal pattern.[12] One band encircles the RC and the other encircles the AB (Fig. 3); the transducer bands are connected

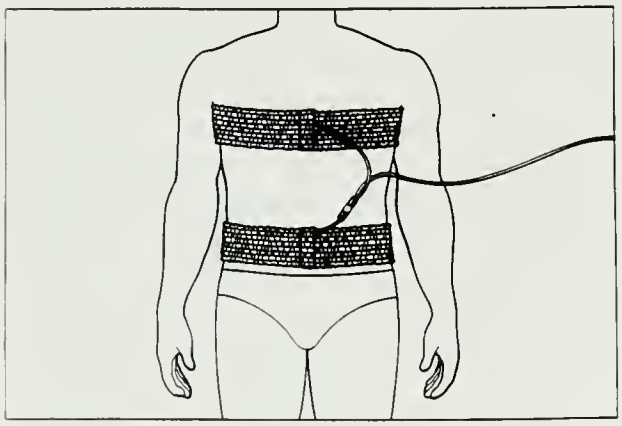

Fig. 3. Proper placement of RIP transducer bands, which are connected by snaps to the oscillator module. The RC band is placed on the upper chest above the breasts in female subjects or at the nipple level in male subjects; the AB band is placed at the level of the umbilicus.

to an oscillator that provides an approximate 20-mV-amplitude sine wave at a frequency of 300 kHz. As the cross-sectional areas of the bands change with respiratory movements, the orientation of the wire coils is altered, thereby changing the oscillatory frequencies as a function of changes in self-inductance. A Z80A-based microprocessor demodulates, amplifies, and converts the analog voltages, which are then displayed on a polygraph recorder or digitized for computer processing.[12] Watson et al[13] utilized 23 separate curved shapes to demonstrate a linear relationship between the cross-sectional area of these models and the output voltage from RIP.

The most recent RIP (Respigraph, NIMS Inc, Miami Beach FL) automatically calibrates the patient during a 5-minute baseline by using the natural breathing, single-posture calibration method,[9] which has been shown to remain accurate for clinical decision making even in the ICU[14,15] despite patient movement.[16] The RIP displays the analog waveforms (Figs. 4 & 5) and reduces the data to a compressed-

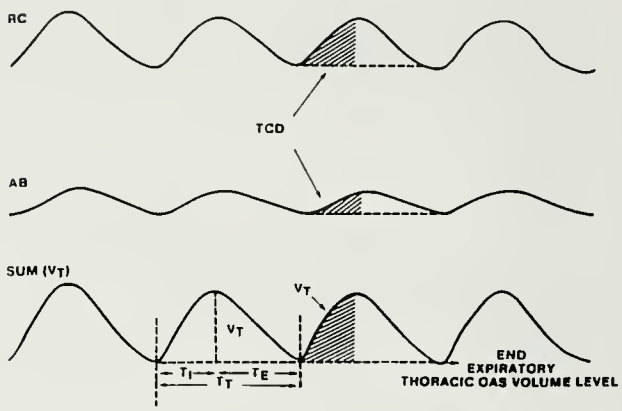

Fig. 4. RIP tracings showing RC and AB waveforms; the electrical sum of the RC + AB = V_T. Total compartmental displacement (TCD) is the absolute sum of the area during inspiration (T_I) of the RC + AB (shaded areas), irrespective of sign. When the RC and AB compartments are in synchrony (as depicted here), the TCD will equal the sum (V_T), and the TCD/V_T ratio will equal unity (1.00). The end-expiratory thoracic gas volume (or FRC level) is shown, as well as timing components (T_E, expiratory time; T_T, total respiratory cycle time).

plot envelope that can be printed (Fig. 6), usually in 15- or 30-minute epochs.

For clinical decision making, the most useful parameters are respiratory frequency (FREQ), V_T (either as an absolute value in mL or as a % change from the calibration period), TCD/V_T (ie, total compartmental displacement/tidal volume, Figs. 4 & 5), %RC/V_T (percent that the RC displacement con-

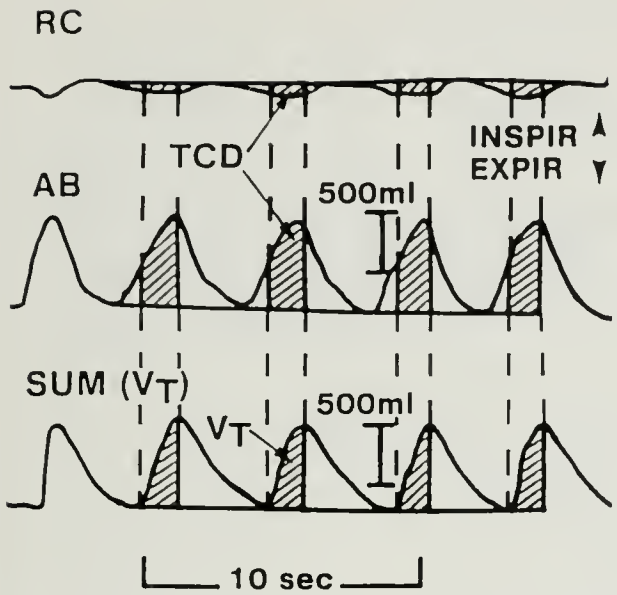

Fig. 5. Analog RIP waveforms from a patient with postextubation laryngeal edema resulting in a partial upper-airway obstruction. The RC waveform moves paradoxically inward or downward during inspiration (INSPIR). Because the RC and AB movements are in opposite directions, the sum (V_T) signal (shaded area) is smaller than the TCD (absolute sum of the shaded areas under the RC and AB—see Fig. 4); therefore, the TCD/V_T ratio is greater than unity (1.91). EXPIR = expiration.

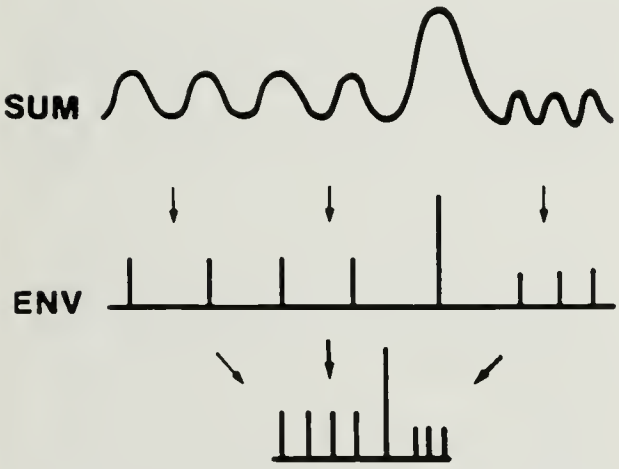

Fig. 6. Illustration showing how the Respigraph system reduces analog breath waveforms to a compound plot envelope (ENV) for convenient record collection. The height of each vertical bar is proportional to the size of the V_T as represented by the sum signal.

tributes to the V_T, Fig. 7), and trend plots of respiratory frequency, V_T, and TCD (Fig. 8).

Applications

Breathing pattern analysis is being used in six areas: (1) the ICU; (1) the intermediate care unit; (3) the sleep disorders laboratory; (4) the pulmonary function laboratory; (5) home monitoring; and (6) pulmonary research. Except for research (more than 400 papers on the RIP alone have been published), the most frequently used applications of ventilatory monitors are in the ICU, intermediate care unit, and sleep disorders laboratory.

Applications in the Intensive Care Unit

Pulmonary catastrophes appear to be heralded by a change in breathing pattern, especially respiratory frequency.[1,15,17–19] The key to effective intensive care is, therefore, close monitoring and recognition of problems before they become catastrophic.[20] Trending of the breathing pattern is extremely useful both during and after weaning a patient from mechanical ventilator support (MVS) as well as when a spontaneously breathing patient may be decompensating. Recent evidence suggests that a major cause of many patients' inability to wean from MVS is relative respiratory muscle fatigue or an inability to keep up with the required mechanical and metabolic demands.[19,20] Although this may not be frank muscle fatigue,[22] breathing pattern changes dramatically in patients who fail to be weaned from MVS[15,17,23] but not in those who are successfully weaned.[14,15,17,23] The hallmarks of developing respiratory muscle failure (Table 2) are increasing respiratory

Table 2. Breathing Pattern Characteristics during the Development of Respiratory Failure

Increasing respiratory frequency (> 11 breaths/min[15])
Thoracoabdominal asynchrony (TCD/V_T increase > 0.22[15])
Respiratory alternans[15,18] or increasing variability of the %RC/V_T[23]
Decreasing V_T[17,18]

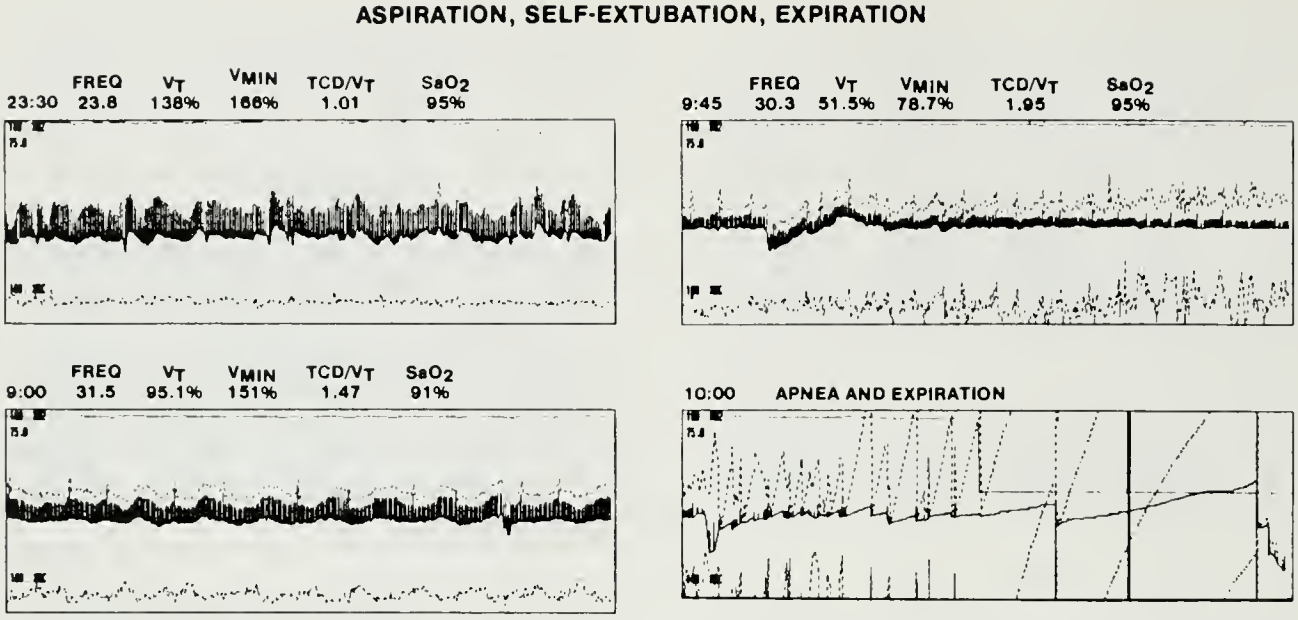
ASPIRATION, SELF-EXTUBATION, EXPIRATION
FREQ VT VMIN TCD/VT SaO2
23:30 23.8 138% 166% 1.01 95%
FREQ VT VMIN TCD/VT SaO2
9:45 30.3 51.5% 78.7% 1.95 95%
FREQ VT VMIN TCD/VT SaO2
9:00 31.5 95.1% 151% 1.47 91%
10:00 APNEA AND EXPIRATION

Fig. 7. Compressed plots from an 85-year-old patient who refused medical treatment. Upper left tracing shows a stable pattern with the patient breathing through a T-piece; note that the %RC is approximately 60% as depicted by the dotted line on the bottom of the tracing. During the night, the patient extubated himself and aspirated (bottom left panel), which resulted in tachypnea (31.5 breaths/min) and an increase in TCD/V_T (1.47) [as shown by the dotted line being above the vertical V_T bars in proportion to the increase in TCD relative to V_T (see Figs. 4 and 5)], which indicates thoracoabdominal discoordination. Over the ensuing 45 min (upper right epoch), the patient's F_{IO_2} was increased, which resulted in improvement in his oxygen saturation as recorded from a pulse oximeter (S_{pO_2}) and displayed at the top of the tracing as S_{aO_2}. However, the V_T has decreased, the TCD/V_T has increased, and the %RC has become much more variable, ranging from > 100% to zero (analogous to "respiratory alternans"). In the final tracing (bottom right), agonal respirations are present; however, S_{pO_2} is relatively stable despite the dying respirations.

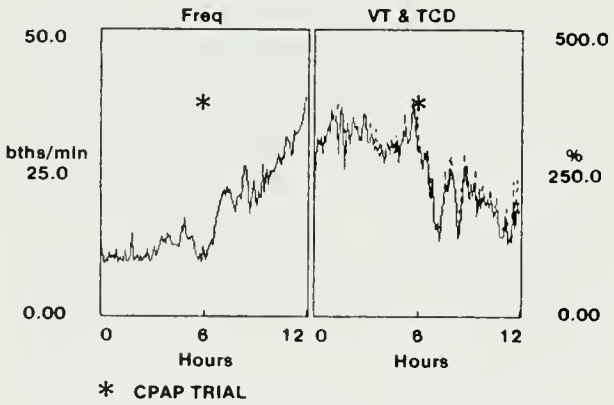

Fig. 8. Twelve-hour trend plots of respiratory frequency (Freq) in breaths/min (bths/min) in the left panel and V_T and TCD on the right. At the asterisk, the patient's physician felt that the patient was ready for a trial of continuous positive airway pressure (CPAP) prior to extubation. Over the ensuing 6 hours, the frequency increased from 10 to 38 and the V_T declined by approximately 100%; these trends are predictive of an unsuccessful extubation.[15,17,18]

frequency,[15,17,18] thoracoabdominal discoordination (increasing TCD/V_T[15,23]); and increasing variability of the %RC/V_T[23] or "respiratory alternans";[15,18] eventually, V_T decreases[18,23] and bradypnea ensues[18] (Fig. 7). Using these criteria, we[15] and others[17,23] have shown a high correlation (up to 99% diagnostic accuracy in our retrospective series[15]) between the presence of these abnormal patterns and the inability to breathe successfully without MVS. However, these are not absolute weaning parameters but rather objective, sophisticated means of trending how the patient looks.

Because of the element of uncertainty associated with the weaning and the eventual removal of a patient from MVS, investigators[24,25] recommend that extubation be performed early in the day when the maximum number of personnel are available to monitor (ie, 'eyeball') the patient. By utilizing four

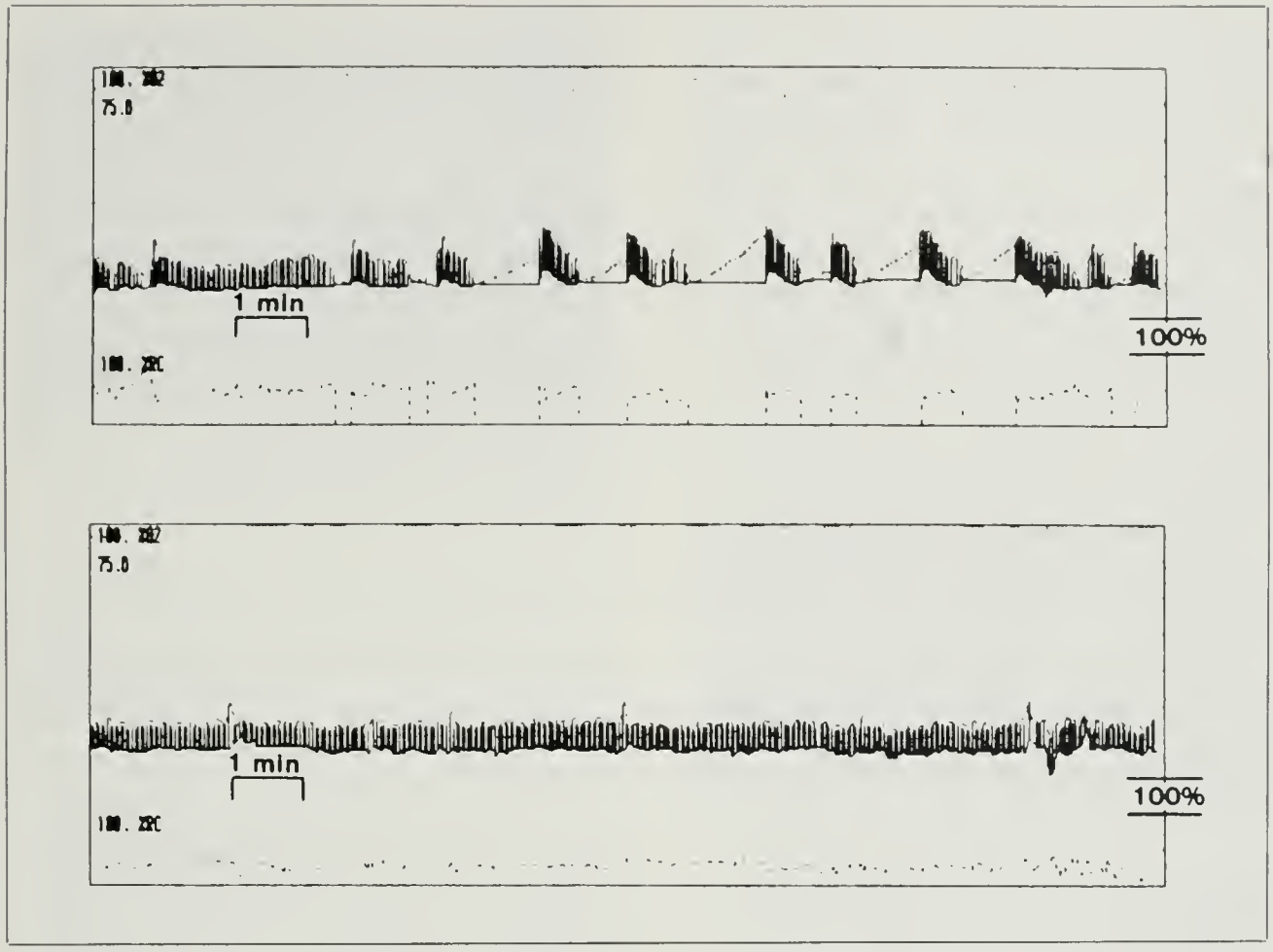

Fig. 9. Fifteen-minute epochs from a 77-year-old patient with a cranial myelopathy following a spinal cord injury that resulted in a functional upper-airway obstruction. The top panel shows an irregular periodic pattern with obstructive events (apneas/hypopneas) lasting up to 57 seconds. The upward sloping dotted line during the apneas is the cumulative TCD and indicates that the RC-AB motion taking place during the events is discoordinate and, therefore, not central; if the RC and AB are not moving and/or are coordinated, the cumulative TCD line would have paralleled the baseline. During the obstructive events, the pulse-oximeter oxygen saturation (S_{pO_2}) recorded at the top of the epoch falls to 67%. The bottom panel shows an epoch from the same patient after a nasal continuous positive airway pressure (CPAP) mask was placed. No events occur and the S_{pO_2} is stable; note the periodic sighs (larger V_T).

self-calibrating RIP units as "substitute nurses" to "observe" the patient, we[26] noted a significant ($p = 0.011$) decrease in the mean (SD) number of hours (compared to the year prior) of patient intubation at our institution [48(4) hours vs 63 (5) hours], with no change in reintubation rates. Continuous monitoring also documented whether the patient was able to adequately sigh and whether techniques designed to promote sighing (coaching, incentive spirometry, intermittent positive pressure ventilation) were effective.

Ventilatory pattern monitoring can also be useful in diagnosing causes of respiratory failure that may occur in the ICU, such as: (1) diaphragmatic flutter;[27] (2) diaphragmatic paralysis;[1-3] (3) upper-airway obstruction (Figs. 5 & 9) or flailed thoracic cage;[1,2] (4) drug overdose; (5) excessive work of breathing secondary to inappropriate ventilator settings;[1,21] and (6) Cheyne-Stokes respirations with central apneas. In addition, simultaneous pulmonary and ECG telemetry allow the staff to distinguish between a cardiac event and a pulmocardiac arrest.[1,2]

Devices placed around the torso measure thoracic gas volume (TGV); at end expiration (Fig. 4), the TGV is the functional residual capacity (FRC). Therefore, intrinsic positive end-expiratory pressure, or auto-PEEP, ($PEEP_I$) can be measured even in spontaneously breathing patients by adding external PEEP until the end-expiratory TGV increases[28] (ie, until FRC is exceeded[29]) as recently shown with RIP (Fig. 10). Because $PEEP_I$ is relatively common[30] and potentially dangerous,[28,31] its easy recognition is important in the mechanically ventilated patient.

Applications in the Pulmonary Intermediate Care Unit

Although cardiac intermediate care units are commonplace, analogous pulmonary noninvasive monitoring units (NIMUs) have been more difficult to establish until recently because of the lack of continuous, reliable, 'user simple' respiratory monitors. By utilizing RIP technology, we established a 4-bed NIMU in October 1986,[32] and have shown significant cost savings (> $3,500.00 per admission[33]), mainly by reducing personnel overhead.[34] Despite the cost reductions, the quality of medical care has been maintained as evidenced by a successful weaning rate of 64% and hospital discharge rate of 58% for the 33 patients who required prolonged MVS (average 26 days). As alternative sites for monitoring patients outside the ICU, units such as NIMUs have the potential to save $500 million annually.[34] In addition, by being used as 'step-down' and 'step-up' units, NIMUs provide a safer[33] environment for moderately ill pulmonary patients who have a high potential for deterioration but do not meet strict ICU admission criteria[35] and for other patients who have the potential to undergo respiratory decompensation (those with neurologic disorders or drug overdoses or those being managed with epidural or on-demand narcotics).

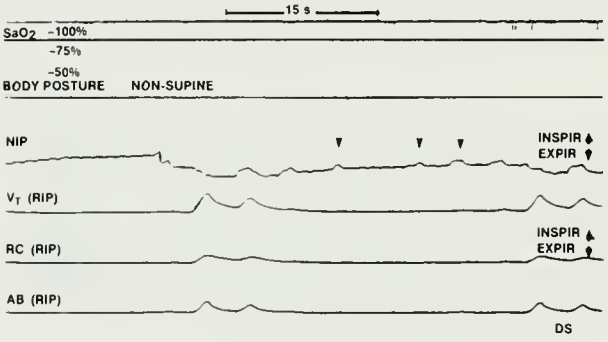

Fig. 10. Polygraph recording from a morbidly obese patient with atypical obstructive apneas. Respiratory inductive plethysmographic (RIP), abdominal (AB), rib cage (RC) and tidal volume (V_T) signals are displayed on the bottom; the neck inductive plethysmographic (NIP) signal, which mirrors changes in pleural pressure, is shown just above the RIP tracings. During the first apnea (as denoted by a flat V_T [RIP] tracing), no NIP movement occurs (except for carotid pulsations). After two large breaths and one hypopnea, the V_T (RIP) signal is again flat; however, the NIP signal shows that efforts are being made (arrowheads) that are not clearly seen on the RIP tracings until the last three breaths. These obstructive efforts were confirmed with an esophageal balloon tracing the following evening.

Other Applications

The most common use of ventilatory monitors placed around the torso is in the sleep laboratory where all the devices listed in Table 1 have been utilized. As noted in the most recent statement from the American Thoracic Society,[36] "there is no doubt that the instrument (RIP) can be used in a qualitative or semi-quantitative fashion to monitor . . . respiratory effort" (Fig. 9). However, "feeble" efforts during obstructive events may be misinterpreted as central events in 5-10% of patients if torso devices alone are relied upon.[37] Placement of an esophageal balloon to record esophageal pressure swings will help detect these small efforts; unfortunately, the technique is invasive and may produce patient discomfort and interfere with sleep quality.[36] Attractive alternatives for noninvasively detecting pleural pressure swings include a surface inductive coil that can be placed over the sternal fossa[38] or—even simpler—a 1-inch inductive band placed around the neck (neck inductive plethysmograph, NIP) that can qualitatively record esophageal pressure swings even when torso devices may be recording a flat signal (Fig. 11).

Devices that can record and store ventilatory patterns have tremendous potential utility for home monitoring as is discussed in a companion paper.[39] However, at the present time, no ventilatory pattern monitor has been shown to be reliable as a diagnostic tool in this setting. Indeed, before use becomes widespread in the ambulatory environment, these devices should be rigorously tested to avoid the potential disasters and unnecessary applications that

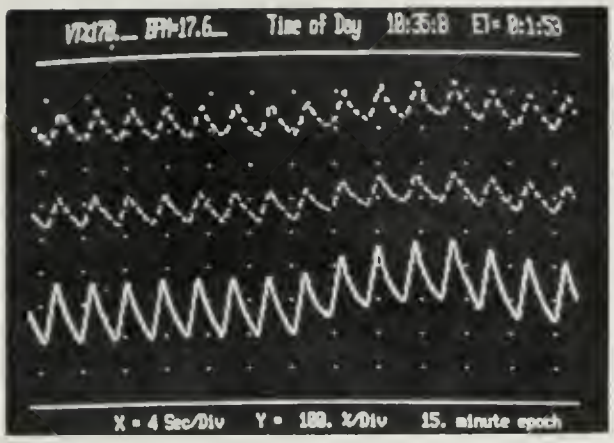

Fig. 11. Intrinsic positive end-expiratory pressure ($PEEP_I$) as measured by respiratory inductive plethysmography (RIP). External PEEP was added to the ventilator every three breaths in increments of 2 cm H_2O. End-expiratory thoracic gas volume (see Fig. 4) increased only after external PEEP was increased from 4 to 6 cm H_2O; therefore, $PEEP_I$ = 5 cm H_2O. Note that the thoracic gas volume returned to baseline when the external PEEP was dropped to 4 cm H_2O at the far right side of the tracing.

have been reported in the SIDS literature,[10] where impedance pneumography was widely used prior to understanding many of its limitations.

Breathing pattern analysis may extend the capabilities of the pulmonary function laboratory, especially when dyspnea is being evaluated. Anxiety reactions[40] and Cheyne-Stokes respirations can be easily documented. In addition, the effectiveness of medication can be followed in this manner.[41] Ventilatory monitoring devices, especially the impedance pneumograph, are also widely used in postoperative recovery rooms, emergency rooms, and endoscopy suites because the analgesics used may precipitate hypoventilation or hypoxemia.[42] Active research using torso devices in infants is also underway[43] in the hope that obstructive events that may have been misinterpreted in patients with SIDS, when impedance devices alone were relied upon,[10] will be more accurately detected (Fig. 5).

Summary

Ventilatory pattern monitoring starts at the bedside with the clinical assessment of the patient. Improvements in noninvasive monitors, especially respiratory inductive plethysmographs, now allow easy monitoring and continuous recording of the breathing pattern. Technology is opening up a new era of understanding that will continue to improve the care of pulmonary patients.

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Krieger Discussion

Chatburn: Bruce (Krieger), what was the inital cost of your monitoring unit? Was it factored into your cost-savings figure?

Krieger: The cost-savings figures included the cost of the equipment amortized over 5 years; it also included an inflation factor of 10%. I wanted the worst scenario, and these are the costs that my administrator came up with—these aren't revenues we generated, these are costs saved.

Hess: If I were to go to my medical vendor to buy a 'Respigraph,' how much would the base unit cost me?

Krieger: The Respigraph has the so-

called QDC calibration; although it sounds technical, it really was the breakthrough needed to move ventilatory pattern monitoring out of the research lab and into the clinical arena. You just press a button and it self-calibrates! The Respitrace unit does not have this capability—you have to do some type of regression or isovolume maneuver to calibrate it. A 4-bed Respigraph unit will cost approximately $100,000. Based upon my figures, if 50% of your hospital patient population has Medicare coverage and your hospital is half filled, the Respigraph will pay for itself in about 9 months. The real cost savings occur if by creating a noninvasive monitoring unit you can reduce the size of your ICU. Thirty to seventy percent of patients in intensive care units don't need intensive care, what they need is intensive monitoring.[1] With the huge cost of ICUs and their personnel, the gross national savings would be tremendous if we could shift patients to intermediate monitoring units.

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Kacmarek: In addition to RIP, are you using pulse oximetry or end-tidal CO_2 monitoring on these patients?

Krieger: We use pulse oximetry on some of these patients. By the time patients are in the final stages of weaning we often don't see a need to continuously monitor S_{pO_2}. We do saturation spot checks. In our noninvasive monitoring unit, we draw on the average one blood gas every other day. We were going to do end-tidal CO_2 monitoring until we did a study[1] in which we found that trends were not useful in clinical decision making. We found minute ventilation to be just as useful a variable to monitor as end-tidal CO_2 or S_{pO_2} (on a continuous basis).

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Kacmarek: My concern is that you've added more monitors than exist in a lot of intensive care units. We send similar patients (after 2 to 3 weeks of intensive care, relatively stable, no longer requiring intensive monitoring) to select general floors where personnel have been specially trained. We're able to maintain the majority of these patients with the alarm systems that are on the ventilators; fewer than 50% of these patients receive ECG monitoring; and few receive oximetry monitoring. We have not had any catastrophes or any significant problems with maintaining these patients under these conditions.

Krieger: What's your nurse-to-patient ratio?

Kacmarek: It's just a general floor—the nurse-to-patient ratio is not altered.

Krieger: I guess I'm working in a different environment in Florida; I think that one of the largest malpractice suits ever settled there was one that involved a ventilator disconnect.

Kacmarek: Yes I know!

Krieger: I know of no hospital in my area of Florida (except the county hospital) where a ventilator goes out to the floor without a nurse-to-patient ratio of at least 1 to 3. And that's where the extra cost comes in. We used to assign 24-hour attendants to each ventilator patient who was transfered to a floor (not that the attendants knew anything about ventilators) just for medico-legal reasons. All we are doing now is substituting a computer (which does not wear out or need to be paid benefits) for the attendants. I do not think that the monitor itself makes the difference! I think it's getting the patients out of the unit that makes the difference! It is easier however, I find, to wean patients when I have a hard copy of what their daily activity looked like. I like to know what happened to my patients, for example, when ventilator changes were made. I can come back 4 hours later and see the trends. In some hospitals with this system, data are not printed out every half hour or every 15 minutes like we do (because we're interested in research); trend plots are just printed out every couple of hours.

East: I was just curious about the QDC calibration technique; I wasn't real clear on how that worked. Could you give us a little more information?

Krieger: The key to the QDC is that you can set the proportionality constant (K) by using isovolume principles and statistical theory during natural breathing.[1] If you thereafter want an absolute volume in mL, all that is necessary is to breath into a spirometer for 4 to 8 breaths. What we use clinically is percent change from baseline—the subject breathes naturally for 5 minutes, which acts as a baseline tidal volume. Everything thereafter is a percent change from that baseline volume. If I'm doing research it takes 4 breaths to set the M term, which proportions the volume of RIP to the spirometer.

1. Sackner MA, Watson H, Belsito AS, et al. Calibration of respiratory inductive plethysmograph during natural breathing. J Appl Physiol 1989;66:410-420.

East: So you've got a built-in multilinear regression technique to compare it to.

Krieger: Yes, I can show you all the mathematics—some of which you'll understand better than I do.

Ritz: Bruce (Krieger), I like the idea of noninvasive monitoring in the sense that it would reduce hospital costs, but the question I have for you is: Was the length of stay in your ICU summarily reduced with the advent of your noninvasive unit or were your number of closed days (where you didn't have admissions) reduced when

you got your noninvasive monitoring unit up and running?

Krieger: The ideal situation would be to reduce not only the number of days, but to actually close beds. I don't think we need more ICU beds, I think we need less! After RIP was introduced into our ICU, the number of hours that patients were on ventilators decreased by approximately 15 hours. However, that doesn't guarantee that these patients actually left the unit any earlier, which is frustrating because we don't control the triage into or out of our ICU. If you send patients who spend an average of 4 weeks on the ventilator out of the ICU, it is the number of mechanical ventilator days in the unit that is reduced. Interestingly, it doesn't mean that the ICU beds remain empty. In our experience, these beds were oftentimes filled with inappropriate cardiac patients who only needed to be in a step-down unit anyway, so it did not work out ideally.

Welch: Could you please tell us what the nurse-to-patient ratio is in your ICU, in your noninvasive monitoring unit, and on your general care floors? Also, how do your patients tolerate the elastic RIP bands around them?

Krieger: I'll take your last question first. We've had very few problems with patients tolerating the smaller bands. In fact, we have just completed a study in which we monitored every patient admitted to our medical intensive care unit (70% of whom were cardiac patients who were awake, alert, and sitting there reading the newspaper). Fewer than 5% had any problems with RIP, as long as they weren't told that it was something new. The nurse-to-patient ratio in our ICU has to be 1:2; in the intermediate care unit it is 1:5.6 (in other words it ranges from 1 to 4 during the day to 1 to 6 at night); on a regular floor the nurse-to-patient ratio is 1 to 8 or 10!

Hess: I'm curious, how many people in the room use RIP, as Bruce (Krieger) uses it, for general monitoring either in the ICU or in a step-down unit? Anybody? (No hands are raised.) A couple for research.

Pierson: We represent 15 different ICUs.

Krieger: I do have a comment about that! I tried to use RIP for research about 6 years ago, but I could never get my program to work correctly. It wasn't until a more user-simple microprocessor was developed that I was able to use it on a continuous basis or on a research basis. It's used most, I think, in sleep laboratories. But other devices are also used in sleep labs, such as bellows pneumographs.

Hess: Is that what Vitalog uses?

Krieger: Vitalog uses inductance circumferential transducers; the figure below shows how the data scattered during change of position.

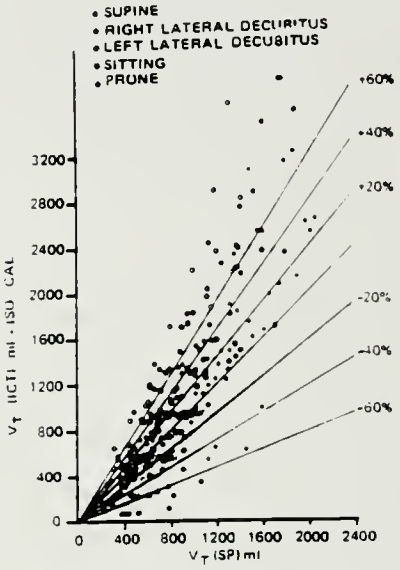

Fig. 1. Identity plots of tidal volumes measured with inductance circumferential transducers [V_T (ICT)] compared to spirometry (V_T (SP)] from six males after changes in posture and V_T. There is a very wide dispersion of V_T (ICT) relative to V_T (SP).

Noninvasive Hemodynamic Monitoring

Charles G Durbin Jr MD

Introduction

Noninvasive monitoring of the cardiovascular system has been an important part of the physician's art for thousands of years. The best diagnosticians of the past noted the character, regularity, and quality of the palpated peripheral pulses to identify disease and to predict the time of death. In 1628, the circulation of blood was described by Harvey,[1] but, it was not until 1733 that blood pressure was directly measured and the pulsatile nature of circulating blood was noted by Hales.[2] Most of the early observations of blood pressure were invasive—made through direct intra-arterial measurements in animals.

In 1876, von Basch[3] developed a technique using a hydraulic cuff to occlude a peripheral artery against a boney prominence; systolic pressure was measured by reducing the intracuff pressure until pulsations were noted in the hydraulic system. In 1896, Riva-Rocci[4] described a technique to measure systolic blood pressure by inflating a cuff (with air) around the arm until the pulse disappeared. Korotkoff[5] is credited with proposing the auscultatory method for systolic and diastolic blood pressure determination (in 1905). This method remains the standard and the basis for current noninvasive blood-pressure monitoring.

Although more difficult to monitor, cardiac output and organ blood flow are physiologically more important to organ function than is blood pressure per se. Therefore, I will describe the currently available methods for the noninvasive monitoring of both arterial blood pressure and cardiac output. When appropriate, noninvasive techniques will be compared to invasive techniques.

Monitoring vs Measuring

The difference between measuring and monitoring vital signs needs to be defined. Monitoring means to measure repeatedly over time. The primary purpose of monitoring is to detect early changes (trends) or to confirm that no change has occurred. What is being monitored may or may not be the variable that is being repeatedly (or continuously) measured; for instance, continuous electrocardiogram (ECG) monitoring is usually provided for the patient hospitalized in a coronary care unit after a myocardial infarction. This is not to detect or diagnose the infarction (a formal 12-lead ECG is used for diagnosis) but rather to detect the development, or progression, or absence, of malignant dysrhythmias requiring treatment. The measuring device that is used to diagnose can also be used as the monitoring device to track the response to treatment.

Most commonly, monitors are evaluated by determining how accurately they measure the variable under consideration. Other equally important characteristics of a monitor include accuracy of trending, ease of use, reliability over time (zero drift and calibration), side-effects (risks), durability, clinical utility of the variable monitored, and cost.

The frequency of measurement that constitutes monitoring varies according to the expected rate of potential change that needs to be detected and the time course of the therapy to be instituted. As an illustration, an automatic device can be used in a physician's clinic to 'monitor' a patient's blood-

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pressure response to oral antihypertensive medication over a period of weeks or months, and during this time changes in dosage are made. The same device can be used to monitor the effect of a potent intravenous vasodilator (eg, nitroprusside) in the coronary care unit, with repeated measurements made every minute. The monitoring requirement in the first case is that the device accurately reflect what would be measured by the Riva-Rocci method (Korotkoff sounds) of blood-pressure determination, which has been used in population studies demonstrating the need for treatment of hypertension. In the second case, the requirement is that the device rapidly detect changes in blood pressure while a vasoactive drip is titrated, the absolute value being of less importance (except in the extreme ranges). The same device should be evaluated as a monitor differently in each situation.

Monitoring Systemic Blood Pressure

First to be considered is blood-pressure measurement. Direct intra-arterial measurement is often taken as the 'reference' standard; however, this is not always appropriate because many factors can alter the directly determined pressure value. Intra-arterial catheters measure a pressure wave that is subject to many mechanical and mathematical distortions. The most important is resonance—both in the external line and in the patient's cardiovascular system.

In general, resonance does not affect average or mean pressure, but it does increase systolic pressure and decrease diastolic pressure. Patient-induced resonance is most likely to occur in the presence of hypertension, tachycardia, atherosclerosis, old age, and small peripheral vessels. The location of the catheter in the arterial tree determines the pressure observed. In Figure 1, the intra-arterial pressure waveform is seen as the catheter is withdrawn from the aortic root to a peripheral artery (in a supine individual). As the distance between the catheter and the central aorta increases, the systolic pressure increases and the diastolic pressure decreases, but the mean pressure remains constant.

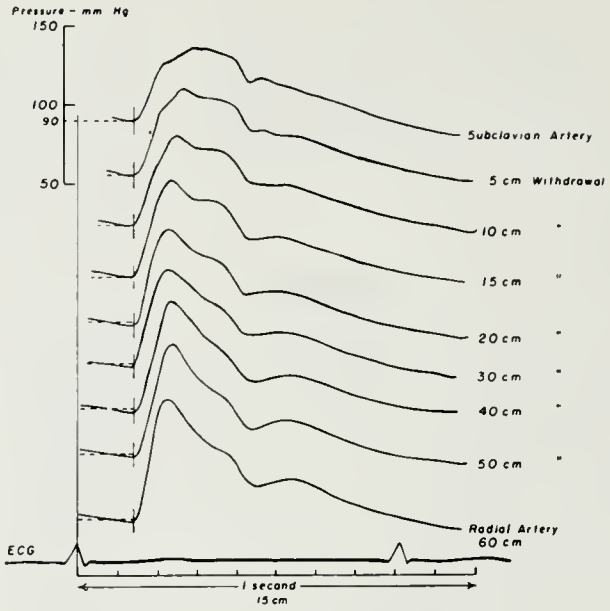

Fig. 1. Arterial pressure waveform changes due to resonance as a catheter is withdrawn from the central aorta into a peripheral artery. (Reprinted, with permission, from Marshall HW, Helmholz HF, Woods EH. Physiological consequences of congenital heart disease. In: Hamilton WF, Dow P, eds. Handbook of physiology. Vol 1. Washington DC: American Physiological Society, 1962:417.)

The same problem with resonance occurs if the transducer is separated from the artery by a length of tubing. The shortest length feasible should be used, and the response of the system should be confirmed prior to accepting displayed values. A lucid description of the concept of resonance is presented in the monograph by Geddes.[6]

Abbreviations Used in this Paper

C_{aCO_2}	— CO_2 content of arterial blood
$C_{(\bar{v}-a)CO_2}$	— Difference between $C_{\bar{v}CO_2}$ and C_{aCO_2}
C.O.	— Cardiac output
CO_2	— Carbon dioxide
$C_{\bar{v}CO_2}$	— CO_2 content of mixed-venous blood
ECG	— Electrocardiogram
P_{CO_2}	— CO_2 tension (pressure)
$\dot{V}_{CO_2}$	— CO_2 production

Noninvasive Methods of Measuring Blood Pressure

Three basically different noninvasive techniques are used to determine blood pressure (Table 1): detection

of flow distal to an occlusive cuff, detection of oscillations in a pressurized cuff, and servo-controlled arterial plethysmomanometry with a partially pressurized cuff. The first two methods require inflation and deflation of the cuff for each determination (intermittent). The third technique provides beat-by-beat (continuous) monitoring after a calibration deflation cycle has been performed.

The classic noninvasive method of blood-pressure determination uses Korotkoff sounds and is called the Riva-Rocci method. Flow is detected by the sounds that result from turbulence in a partially occluded artery. The sounds start as flow begins (the systolic point) and become muffled or disappear as the vessel becomes undistorted by the cuff (the diastolic point).

The arterial pressure is the cuff pressure at which these changes in sound occur. Inaccuracies in measurement can occur if the cuff is too large (low measurement) or too small (high measurement). The proper cuff width is 120 to 150% of the extremity diameter.[7] The appropriate cuff-bladder size is one half to two thirds of the circumference, and the cuff must be snugly applied but must not be occlusive. In all cuff-based techniques, these same morphometric considerations should be kept in mind.

Table 1. Noninvasive Methods of Determining Blood Pressure

	Manual/Automatic
Distal Artery Flow Methods	
Palpation	
Riva-Rocci	M
Oscillotonometer	M
Auscultatory	
Korotkoff Sounds	M
Doppler methods	
Arteriosonde	A
Arterial Line Flow	M
Photoelectric	
Pulse oximeter	M
Doppler Flow Probe	M
Oscillometric Methods	
Dynamap	A
Servo-controlled Plethysmomanometer (Peñaz)	
FINAPRES	A

Another source of inaccuracy in cuff-pressure methods is that tissues do not necessarily distribute the pressure equally. In a severely sclerotic vessel, much of the cuff pressure required to occlude blood flow may be needed to compress the vessel rather than overcome the arterial pressure. Arterial pressure will therefore be overestimated. The presence of other hard structures such as bone or tendons may also affect the distribution of pressure in the artery.

Table 1 lists the other flow methods of blood-pressure determination. The 'return to flow' techniques require detection of the pulse distal to the cuff and are generally useful only for determination of systolic pressure.[8] The pulse can be detected with a finger, a Doppler flow detector, a pulse oximeter, or an indwelling arterial line. (Only the presence of a pulse trace is needed to determine the pressure.)

To determine systolic pressure with these techniques, the pulse detector is appropriately placed and its function confirmed. The cuff is then inflated and gradually deflated. At the first detection of a pulse, deflation is stopped and the pressure determined—this is the systolic point. It is supposed that no diastolic pressure can be accurately determined by these methods.

The systolic pressure determined by these methods correlates well with that determined by the auscultatory method, but it is usually lower than that obtained by intra-arterial pressure determination.[9,10] These are manual methods used to quickly check arterial pressure or to confirm the accuracy of other automatic methods. They are rarely used as monitors in patients with rapidly changing conditions.

The oscillotonometer uses a special cuff with two bladders. The upper bladder is the occluding bladder, and the pressure difference between the two bladders is displayed in oscillations of the manometer needle. When oscillations begin, systolic pressure is determined; when they cease, diastolic pressure is determined. This device overestimates low systolic pressure and underestimates high systolic pressure.[11] Accuracy of diastolic pressure determination is very poor.[12]

The first automated-flow method for blood pressure determination was the Doppler-based Arteriosonde (Kontron Instruments, Everett MA, formerly manufacturered by Roche, Hoffman, & Laroche, Cranbury NJ).[13] By detecting the movement of the arterial wall during automatic cuff deflation, this

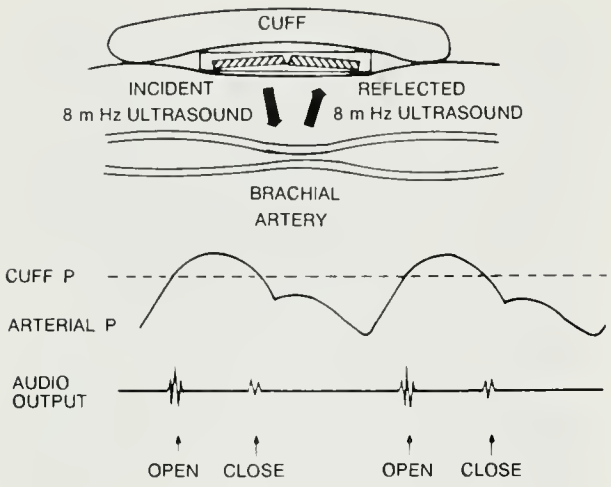

Fig. 2. The method of using ultrasound to 'hear' vessel wall movements during automatic cuff deflation (Arteriosonde). (Reprinted, with permission, from Reference 6.)

device is able to identify systolic and diastolic pressures. As illustrated in Figure 2, the ultrasound Doppler probe is placed under the cuff and directly over the artery. The signal is transmitted toward the vessel and only if the wall is moving does a Doppler shift occur in the receiver. Because the slightest displacement of the ultrasonic crystal alters the signal, it is necessary for the patient to be absolutely still for this monitor to function consistently.

Oscillometric Methods of Blood Pressure Determination

Erlanger investigated the pressure changes or oscillations that occur during cuff deflation.[14] The maximum oscillations occur shortly after systolic pressure and the last maximal oscillation occurs at diastolic pressure. With appropriate mathematical corrections and the discovery that maximal oscillation occurs at true mean arterial pressure, this method provides the basis for many currently used automated blood-pressure devices. An average arterial pulse trace is constructed from the amplitude of oscillations (Fig. 3), and a calculation of systolic and diastolic pressure is made. Repeated determinations can be made with these devices as frequently as every minute. Accurate systolic values can be obtained in hypotensive patients, but accuracy is lost (blood pressure is underestimated) in hypertensive patients.[15,16] Diastolic pressure determinations are less accurate. A significant risk posed by the potential tourniquet effect of these automatic devices is tissue ischemia.[17] Awake patients complain of pain or discomfort if the device is used for a prolonged period of time or for short intervals on a frequently repeated basis. However, these devices can be effective monitors when changes are not expected to occur more frequently than every few minutes. These devices are subject to the same cuff-to-extremity size limitations as are the manual inflation devices.

My personal concern about such devices is that no manual way exists of confirming the accuracy of the pressure determined by these devices. It is not possible to determine a systolic pressure by palpation using the same cuff. Because these devices are sensitive to motion artifacts, it is not uncommon for erroneous values to be displayed. The cycle time can be quite long especially when cardiac rhythm is irregular (as in atrial fibrillation). I have observed that a readout of zeroes can mean either a mechanical problem with the device or a very low blood pressure in the patient. As machine failure becomes common, attention may be shifted to the device instead of confirming the

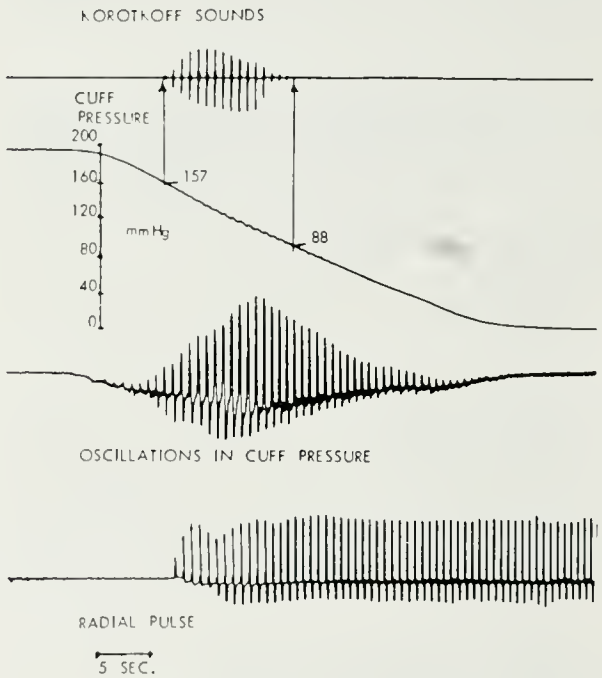

Fig. 3. Simultaneous recording of return to flow (arterial line), cuff oscillations, and Korotkoff sounds during cuff deflation. Maximum oscillations occur at mean arterial pressure. (Adapted, with permission, from Reference 6.)

vital status of the patient. Newer models have a 'stat' mode for rapid-cycle manual pressure determination in questionable situations. A power source, either battery or line current, is necessary for these devices to function.

Servo-Controlled Constant Artery-Volume Blood-Pressure Determination

In 1972, Peñaz[18] proposed a continuous noninvasive method of infrared detection and rapid plethysmographic servo-controlled arterial-volume measurement of finger blood pressure. The FINAPRES (Ohmeda, Madison WI) is one device that uses this method; the FINAPRES automatically determines the volume of the digital arteries when the pressure inside equals that outside (Fig. 4). Then, in this unloaded condition, a servo pressure-controller prevents enlargement of the arteries that are surrounded by the cuff. The pressure needed in the finger cuff is identical to the intra-arterial pressure, and the waveform is displayed.

The values obtained by this device seem to underestimate values obtained by intra-arterial determination by 2 to 3.5 kPa.[19] In a comparative study, the FINAPRES provided values that more closely approximated intra-arterial pressure than did an oscillometric device. The FINAPRES also provided a continuous arterial pressure tracing.[20] The continuous application of pressure to the fingertip can result in venous occlusion and ischemia.[21]

A similar principle has been applied to arm cuffs, and similar continuous arterial pressure tracings have been produced. Verification of this arm-cuff device, the Cortronic 7000 (Cortronic Corp, Ronkonkoma NY), awaits clinical experience. The venous occlusion with this system may be clinically important. These new devices have the potential of replacing invasive monitoring because they are instantaneously responsive to changes in blood pressure, they have a low complication rate, and difficulty in identifying equipment failure is not encountered.

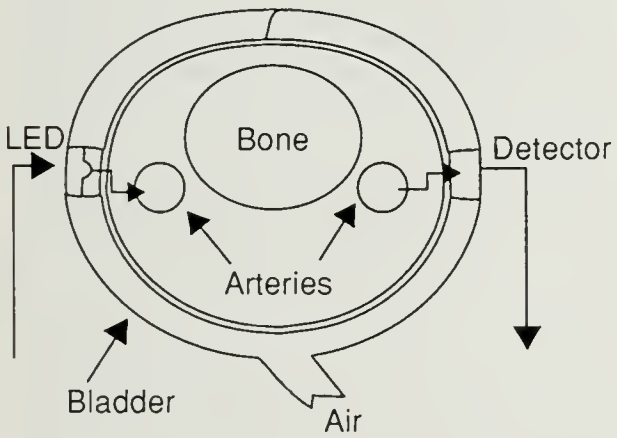

Fig. 4. Cross-section of a finger with the cuff and photodetector positioned. A servo-controlled device applies pressure to the bladder to maintain constant output from the detector. The pressure required to maintain light path of constant length is the arterial pressure waveform. (Reprinted, with permission, from Boehmer RD. Continuous, real-time, noninvasive monitoring of blood pressure: Peñaz methodology applied to the finger. J Clin Monit 1987;3:283.)

Noninvasive Determination of Cardiac Output

The standard method to determine cardiac output (C.O.) is the invasive indicator-dilution method (based upon the Fick principle).[22] Indocyanine green[23] or suitable cold solutions such as normal saline are injected through a thermistor-equipped pulmonary artery catheter[24] to determine C.O. The usefulness of C.O. measurements and hemodynamic monitoring is well established.[25] Noninvasive methods to determine C.O. include changes in thoracic electrical impedance, aortic Doppler flow methods, and CO_2 rebreathing (indirect Fick method).

When an alternating current is applied to the thorax, the impedance changes depend on the distribution of blood. Because the distribution of blood in the chest changes as blood volume of the heart changes (the path of least electrical resistance), varying with systole and diastole, the impedance changes are directly related to cardiac stroke volume. Several complicated formulas have been used to calculate stroke volume. Included in the formulas are the distance between the electrodes, hematocrit, left-ventricular ejection time, and body temperature.[26] This calculated stroke volume is multiplied by the heart rate to give C.O.

The correlation between the value from the impedance cardiograph and values by other methods has been variable. In healthy volunteers, animals, and stable patients with cardiac disease, impedance

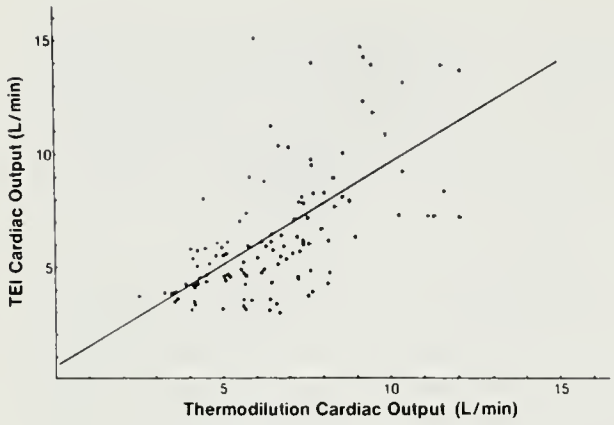

Fig. 5. Concurrent estimations of cardiac output by thermodilution (abscissa) and transthoracic electrical impedance (TEI). The line of linear regression is shown (r = 0.63, y = 0.35 + 0.93x). (Reprinted, with permission, from Reference 30.)

cardiography reflects the changes (but not the exact values) identified by other methods.[27-29] Less useful results have been obtained in critically ill populations. In a study of 27 critically ill patients, the correlation between thermodilution determinations and impedance methods was only 0.63 (Fig. 5).[30] Both over- and underestimation occurred. Technical difficulties in maintaining electrode placement and carotid upstroke recording (to determine left-ventricular ejection time) were frequently encountered with these patients. No correlation between impedance and wedge pressure could be demonstrated. No other hemodynamic variables can be derived from this method (eg, stroke work, resistance, central venous pressure, mixed-venous oxygen saturation). The authors concluded that the electrical impedance determination of C.O. is unsatisfactory for monitoring critically ill patients, although it did appear valuable for monitoring stable patients (eg, in the exercise physiology laboratory).

Although the actual results obtained by this method may not truly represent C.O., the method can be used continuously and may accurately reflect trends. This feature fulfills one of the definitions of a monitor; therefore, this method should be viewed from that perspective. Data on trending, ease of use, reliability, and costs are not yet available.

Doppler methods can be used to determine C.O. noninvasively. Such methods depend on our knowing or measuring the aortic diameter. The average flow across the aortic cross-section is then measured, and stroke volume is calculated. C.O. = stroke volume × heart rate. The accuracy of measurement of the velocity vector depends on the maintenance of a precise, optimal angle to aortic flow (Fig. 6). The Doppler probe can be placed in an extrathoracic (transthoracic),[31] transesophageal,[32] or transtracheal[33] position.

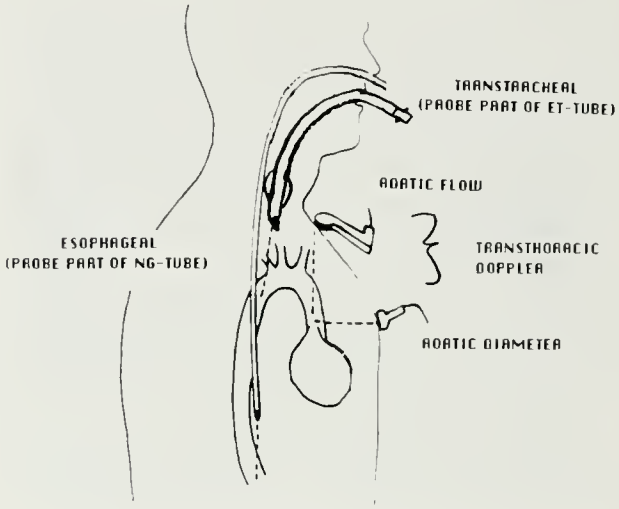

Fig. 6. Location of Doppler flow probes for cardiac output measurement. Knowledge of the aortic diameter (measured with Doppler or estimated by formula) is needed for measurement. Transthoracic (including aortic-root-diameter determination), esophageal, and transtracheal locations are illustrated.

All of the Doppler techniques depend on the location, signal direction, and acoustic contact of the Doppler ultrasound probe. User skill and experience are the keys to reproducible results. Movement of the probe is responsible for many of the changes seen, and artifacts are not easy to differentiate from actual cardiac function changes. Comparison with other methods has shown variable correlation even in stable healthy patients.[33] These devices are large, delicate, and expensive.

Another way to determine C.O. is with a pseudo-steady state carbon-dioxide-rebreathing technique (the indirect Fick method).[34-37] C.O. is calculated using the equation:

$$C.O. = \dot{V}_{CO_2}/C_{(\bar{v}-a)CO_2},$$

where $\dot{V}_{CO_2}$ is CO_2 production and $C_{(\bar{v}-a)CO_2}$ is the difference between mixed-venous CO_2 content

($C_{\bar{v}CO_2}$) and arterial CO_2 content (C_{aCO_2}). $\dot{V}_{CO_2}$ is measured continuously; C_{aCO_2} is derived from arterial P_{CO_2}, which is calculated from end-tidal P_{CO_2}; and $C_{\bar{v}CO_2}$ is derived from mixed-venous CO_2, which is estimated from a 15-second period of rebreathing 10% CO_2 (intermittently performed). A number of automated, microprocessor-driven metabolic carts are now capable of measuring C.O. noninvasively using the CO_2-rebreathing technique. In healthy, cooperative, unintubated volunteers, this method has been shown to be more accurate in determining C.O. than the bioimpedance technique.[38] However, further work is needed to establish the reliability and usefulness of this method in clinical practice.

In summary, several promising methods have been developed for determining C.O. noninvasively; however, they await confirmation of clinical usefulness. In general, these noninvasive methods are less accurate than are invasive methods, but they are valuable because C.O. determination can be made repeatedly to provide continuous monitoring.

Conclusions

Several alternatives to invasive hemodynamic monitoring exist. The stability of the patient, the expected rate of change, the needed information, and the balance of risks determine the method to be used. An algorithmic approach to choosing the appropriate method is presented in Figure 7. If the patient's clinical situation is stable and if the hemodynamic changes are expected to occur slowly (as in a patient beginning antihypertensive therapy at home for chronically elevated diastolic blood pressure), noninvasive blood-pressure measurements performed each week by either the auscultatory technique or an automated oscillometric device (for convenience) are appropriate—certainly safe and cost-effective.

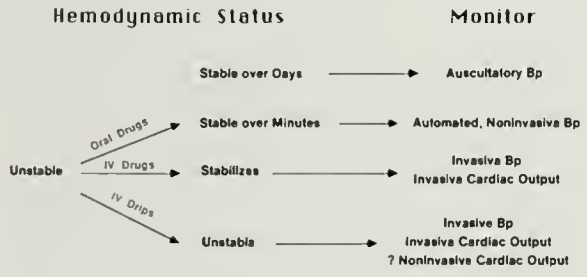

Fig. 7. An approach for selection of the appropriate hemodynamic monitor.

In an otherwise healthy patient who is experiencing acute blood-pressure changes (eg, while under anesthesia or while receiving sublingual nitroglycerin), more frequent measurements are needed and an automated system is desirable. In critically unstable patients who may be experiencing blood-pressure changes every two or three heart beats (eg, while receiving intravenous vasoactive drips such as epinephrine or nitroglycerin), or in whom very low pressures are likely (eg, in septic shock), continuous monitoring is needed. This may be provided by the continuous noninvasive techniques (eg, FINAPRES); however, invasive arterial monitoring is superior and allows for the sampling of blood for gas and electrolyte analysis.

The role of noninvasive C.O. monitoring has yet to be defined. Noninvasive techniques offer the advantage of continuous monitoring; however, because of their inherent inaccuracy and inability to measure other hemodynamic variables, it is unlikely that they will replace invasive techniques. In stable, chronically ill patients they provide an otherwise unavailable measurement and thus may contribute to a better understanding of disease pathophysiology at reduced risk to the patients.

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Durbin Discussion

Hess: We've played around a little bit with noninvasive cardiac output in our ICU. In fact, we even made the mistake of buying one of the Doppler units and have concluded that it doesn't work very well. The other thing that created a problem was that we weren't really sure when it would be useful to know what the cardiac output was without knowing what the wedge pressure was. So, what seemed to happen was that when we wanted to know the cardiac output, we also wanted to know the wedge pressure; and if you need to get a wedge pressure you need to put in a Swan-Ganz catheter, so then you can do a thermodilution cardiac output. Do you have thoughts on resolving that issue?

Durbin: The same questions arose; I have a Doppler device that we paid $25,000 for that's gathering dust in the corner.

Hess: Do you want to put them together?

Durbin: I tried to trade it in—on a Mercedes, anything! It seems to work best in preoperative evaluation—for instance, if you want a rough estimate of how much functional cardiac reserve there is. It doesn't work well postoperatively because the anatomy, at least in cardiac surgery patients, is altered by sternotomy and lung changes. Patients on ventilators, for instance, are notoriously poor candidates for that kind of device. So, I believe that, except for the operating room where continuous monitoring can be done with transesophageal echocardiography, the Doppler probably has no role in the ICU. However, the other devices may, and their continuous nature and ability to reflect trends may be useful. Certainly if you need to know wedge pressure, which you almost always want in a critically unstable patient, then I think you probably need both of them—not one. Right now, continuous invasive cardiac output monitors are not available, although there are some that are in developmental stages.[1,2] That might be the way to go rather than to go with a noninvasive system that really doesn't have that other variable.

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Krieger: Dean (Hess), I'd like to play devil's advocate with you. I agree that a lot of us have grown up thinking that the wedge pressure is a meaningful number that reflects something physiologic.[1] However, I have a certain feeling that Eugene Robin might be correct.[2] The wedge pressure is just a number—one third of the time being a wrong number, or being inaccurately calibrated, or inaccurately measured, or reflecting something other than intravascular preload. What you really are looking for in the critical care area, I think, is more the end result of what is happening and not the factors going into it. In other words, if you're adding volume to a patient, you don't want the wedge pressure to go up—you want to see cardiac output improve. If you're adding pressor agents you don't want to know what's happening to the wedge pressure, you want the final outcome—what you want to know is whether stroke volume increased and/or blood pressure increased. Now, I'm not speaking of diagnosing whether pulmonary edema is cardiogenic or noncardiogenic. I'm talking about using the wedge pressure as a monitor for clinical decision making. I think a lot of us have lost sight of some basic clinical skills that were well known in cardiology 20-25 years ago (before echocardiograms took over). A lot of data can be gained just by looking and palpating the chest and pulse or using devices like the cardiokymogram[3] or the kinetocardiogram. These utilize small transducers placed over the apex of the heart that transmit what looks like a ventricular volume curve. We've actually been using a 1-inch-wide inductive plethysmographic band placed just below the xiphoid. When we exclude respiration, which is very easy to do with microprocessors, we've been seeing curves nearly identical to ventricular volume curves. So, I do think that there is a role for noninvasive cardiac output or stroke volume measurements. They don't have to be in absolute terms—I'm used to thinking of things as %changes from baseline.

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Durbin: Along those same lines, I think that the use of any of these numbers is more relevant in terms of the change in that number after an intervention than it is in the absolute number. And that's why calibration may not be the key to monitoring (as you say) with any device. Whether it's left-ventricular end-diastolic volumes or wedge pressures—it really isn't important! What is important is the change after an intervention—that gives much more information. Most monitors have never been looked at in terms of trends because it is so difficult to do. It's difficult to look at whether a trend analysis helps; I think we all know it does, but we can't prove that in a scientifically rigorous way—we can certainly know whether the number is

the same as that which we get with another device.

MacIntyre: Charlie (Durbin), what do think of soluble gas techniques that have been around for years that use diethyl-ether or acetylene during a rebreathing maneuver—you can get not only cardiac output (or at least pulmonary capillary blood flow) but also, because of its biexponential decay, you actually pick up the variable called tissue volume or lung water? My own prejudice is that while this may be useful in reasonably healthy lungs (eg, in exercise facilities), there may be too much noise in ICUs. Do you have any comments?

Durbin: If you look at how a monitor becomes useful, one of the ways is that it has to be easy to use. The devices that I've seen aren't really that easy to use. It's a whole lot of equipment and reproducibility in the laboratory is very difficult to obtain—at least with multiple gases. Maybe with one or two it might not be as much of a problem. If the soluble gas techniques were easily available, we would all be using them to measure FRC, as Dave (Pierson) pointed out this morning. It has to be easily available, it has to have some clinical conceptual uses, it has to work, it has to be at the bedside, and you have to be able to use it to see if it's going to actually help you manage patients better. I don't think monitors really do that much for us—all they do is extend what we already know, they only extend our senses a little bit! They may show us something in a different way, but they really don't change what's wrong with the patient. They have to prove themselves in the clinical environment. If they're useful and clinicians find them helpful, everybody will have one. If they're too complicated, not durable enough, or too expensive—nobody will have them. That's not answering your question. Sell me one and I'll try it! [laughter]

Marini: This is perhaps a little unfair Charlie, but I was wondering if you could help me out on the use of invasive hemodynamic monitoring to monitor respiratory events. There used to be quite a lot of enthusiasm for looking at fluctuations in CVP, or wedge pressure, or the like to monitor intrathoracic pressures. Most of us are not anxious to put esophageal balloons into our patients, but we often have (in patients with ARDS anyway) a Swan-Ganz catheter in place from which you can get both pressures. Is there any consensus about using that kind of information for respiratory monitoring?

Durbin: I think anecdotally we use it—I use it on rounds to demonstrate particular findings, but I don't know that there is any systematic approach to using that data. I use it anecdotally when it occurs, but I don't really seek it. It's sort of like I already know what's wrong, and therefore I find it, as opposed to "Ah! There it is, I need to worry about it."

Welch: Over the past four years we've collected a large hemodynamic patient database on analog tape, including multiple leads of ECGs and multiple pressures as well as airway, CO_2, and impedance plethysmography. We have found that the CVP trace is more sensitive than the impedance signal for recovering respiratory rates, and, in fact, if you look at the CVP trace you can pick out the type of ventilation, whether it be controlled or spontaneous. Unfortunately, there aren't algorithms implemented in bedside monitors to take advantage of that signal, but the information is certainly there and it compares very favorably with CO_2 monitoring for respiratory rate.

East: To follow up a little bit on Dr MacIntyre's question, I know companies are thinking about rebreathing gas techniques—whether it's acetylene, CO_2, or whatever—because they're attractive (they can be implemented in a fairly easy-to-use package). The problem it always comes down to is—Is effective pulmonary blood flow of interest? I don't think you're going to get reliable cardiac output out of it. A question that I have for you or anyone else here is: Would a device that could measure effective pulmonary blood flow be of clinical usefulness?

Durbin: I guess the answer is "no."

Krieger: Charlie (Durbin), have you used systolic time intervals at all for following cardiac output or stroke volume?

Durbin: My experience is fairly limited. When I first came to Virginia 10 years ago, that was the 'going' concept. In fact, one of the folks there developed a computer program for doing that sort of thing on-line. It, like all other ECG-derived variables or ejection-derived variables, has many reasons for changing. It didn't pinpoint any particular abnormality that we could follow (that we could focus in on); we knew when things were bad, but we didn't really know what to do next. And, even though the systolic time interval was reasonable, it didn't mean the cardiac output was good. We did not find it to be very helpful.

Krieger: I had a similar experience when I used it to look at single points in time, but I wonder if as a trend monitor it might prove to be useful?

Noninvasive Monitoring of Respiratory Function Outside of the Hospital

Robert M Kacmarek PhD RRT

Introduction

The practice of providing respiratory care in the home developed rapidly during the 1980s. At present (1990), oxygen therapy, mechanical ventilation, and bronchial hygiene procedures are performed regularly in the home care setting. However, little information is available to guide the respiratory care practitioner in the appropriate use of noninvasive monitoring in the home. Most practitioners involved in home care realize that *the home is not an alternate site for intensive care* and that respiratory care techniques applied in the home need to be simple and safe. As a result, the need for continuous noninvasive respiratory monitoring in the home is limited. Apart from respiratory mechanics monitoring and general patient assessment, only pulse oximetry and end-tidal carbon dioxide (et_{CO_2}) monitoring have been used in the home care setting.

Noninvasive respiratory monitoring techniques are used extensively in apnea screening and monitoring; in fact, because of the backlog in many sleep laboratories, home screening of apnea is becoming the preferred method of initial evaluation.

Abbreviations Used in this Paper

CPAP	— Continuous positive-airway pressure
F_{IO_2}	— Fractional inspired oxygen concentration
$etCO_2$	— End-tidal carbon dioxide
P_{aO_2}	— Arterial oxygen tension (partial pressure)
P_{etCO_2}	— End-tidal CO_2 tension
S_{aO_2}	— Arterial oxygen saturation
S_{pO_2}	— Oxygen saturation measured by pulse oximetry

Noninvasive Monitoring of Oxygen Therapy

Oxygen therapy is the single largest element of respiratory home care. Determination of need for oxygen therapy has essentially been based on data from the Nocturnal Oxygen Therapy Trial[1] and the British Medical Research Council Trial.[2] Both of these studies demonstrated decreased mortality and improved physiologic function with the long-term use of oxygen therapy. Based on this data, reimbursement criteria for oxygen therapy by Medicare were established.[3] A maximum arterial oxygen partial pressure (P_{aO_2}) of 55 torr or an arterial oxygen saturation (S_{aO_2}) of $\leqslant$ 85% breathing room air were originally necessary for reimbursement. However, an S_{aO_2} of 85% under normal physiologic conditions (pH 7.40, P_{CO_2} 40 torr, temperature 37 °C) does not represent a P_{aO_2} of 55 torr but one of 52 torr. Thus, based on these original criteria (Table 1) the use of S_{aO_2} alone to establish need for O_2 therapy results in the denial of O_2 therapy reimbursement to subjects who would have been eligible if P_{aO_2} had been measured. This fact was dramatically demonstrated

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Table 1. 1985 Medicare Reimbursement Guidelines for Oxygen Therapy*

$P_{aO_2} \leq 55$ torr
$P_{aO_2} \leq 59$ torr and dependent edema or cor pulmonale or hematocrit $\geq 55\%$
or
$S_{aO_2} \leq 85\%$
$S_{aO_2} \leq 89\%$ and dependent edema or cor pulmonale or hematocrit $\geq 55\%$

*In 1989 the S_{aO_2} criterion increased to 88%.

by Carlin et al[4] who compared saturations measured by pulse oximetry (S_{pO_2}) and P_{aO_2} in 55 patients with chronic lung disease. They found that 80% of the patients with a resting $P_{aO_2} \leq 55$ torr had an S_{pO_2} of $> 85\%$ (Fig. 1). As a result, 44 patients in this study would have been denied access to oxygen therapy based on oximetry data alone. This study prompted changes in the Medicare guidelines for reimbursement.[5] Now an arterial saturation (S_{aO_2} or S_{pO_2}) $\leq 88\%$ establishes the necessity for home oxygen therapy. However, data reported by Carlin et al[4] indicate that at a saturation threshold of 88% some individuals who would meet the P_{aO_2} criterion do not meet the saturation criterion.

The value of data from pulse oximetry for the purpose of establishing the need for home oxygen therapy must be further questioned based on the accuracy of these instruments. Studies of individual oximetry units have established the 95% confidence interval for agreement with CO-oximetry at $\pm$ 4.0-5.0%[6-9] at saturations of $> 70\%$. The choice of oximeter, as illustrated by Carlin et al,[4] may also affect the meeting of reimbursement criteria. The Biox IIA pulse oximeter identified a larger number of patients with an S_{pO_2} of $> 85\%$ with a corresponding P_{aO_2} of < 55 torr than did the Hewlett-Packard HP47201A ear oximeter. This variability in accuracy of pulse oximeters was dramatically demonstrated by Severinghaus and Naifeh.[10] At an average S_{pO_2} of 55 $\pm$ 7.5% by CO-oximeter established in volunteers breathing low F_{IO_2} mixtures, errors between CO-oximeter and the 6 pulse oximeters evaluated varied from -7.9 $\pm$ 5.8% to 13.1 $\pm$ 12.7%.

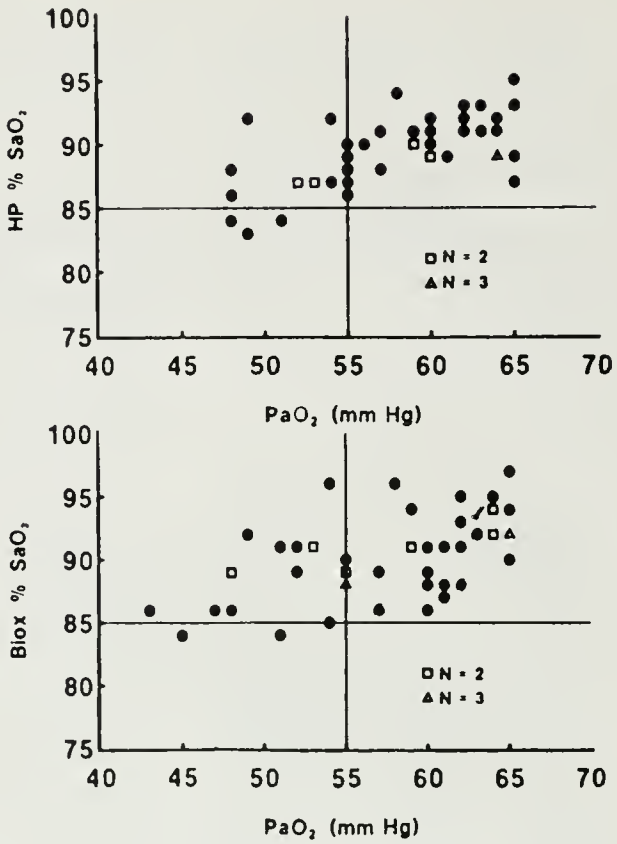

Fig. 1. The relationship between resting arterial oxygen tension (P_{aO_2}) and arterial oxygen saturation (S_{aO_2}) measured by a Hewlett-Packard HP 47201A (top) or a Biox IIA (bottom) oximeter. Solid lines represent 1985 Medicare reimbursement criteria (P_{aO_2} 55 torr, $S_{aO_2} \leq 85\%$). Upper left-hand quadrants represent subjects meeting P_{aO_2} guidelines but failing S_{aO_2} levels. (Reprinted, with permission, from Reference 4.)

This does not imply that there is no place for pulse oximetry in long-term oxygen therapy. It does, however, exclude the use of pulse oximeters as a reliable and accurate means of establishing the need for oxygen therapy. However, once need has been established and the P_{aO_2} for a given individual correlated to a specific S_{pO_2} by a given pulse oximeter, pulse oximeter data are useful in trending change in status or in identifying desaturation during exercise.

No guidelines exist to define circumstances in which continuous pulse oximetry is indicated in patients receiving home oxygen therapy. If we assume that the patient is breathing spontaneously, no rational, cost-effective, medically reasonable argument can be made for continuous oximetry in either adults or infants. All patients at home on O_2 therapy should

be medically stable and, as a result, should not require this continuous monitor. In fact, the use of continuous pulse oximetry may be counterproductive because patients may experience momentary minor swings in saturation during coughing or periods of physiologic stress. The continual or recurrent sounding of an alarm during these clinically insignificant episodes simply increases anxiety in the patient and caregivers, with no beneficial result.

During periodic home visits by health care workers to assess the overall status of patients dependent on oxygen therapy, 'spot checks' with a pulse oximeter seem reasonable, provided P_{aO_2}-S_{pO_2} correlations have been established. This information, coupled with clinical assessment data (Table 2), is useful in identifying deterioration or improvement in status. After hospital discharge, many patients with appropriate medical treatment continue to demonstrate improvement in lung function. As a result, the oxygen prescription may be modified based on such trending information.

Table 2. Considerations in Monitoring of Oxygen Therapy in the Home during Periodic Visits

Recording of results of pulse oximeter spot check*
Recording of results of auscultation, palpation, and percussion of chest
Observation and recording of vital signs
Observation and recording of ventilatory pattern
Observation and recording of sputum volume, color, and consistency
Observation and recording of patient's appearance and presentation

*Continuous pulse oximetry is never indicated.

Noninvasive Monitoring of Positive-Pressure Breathing Therapy

As with oxygen therapy, greater and greater numbers of patients are receiving some level of positive-pressure breathing therapy in the home. This may be positive-pressure ventilation, periodic intermittent positive-pressure breathing treatments, or continuous positive-airway pressure. The level of monitoring required depends to an extent on the number of hours per day support is required and on whether support is elective.

Elective Positive-Pressure Therapy

Greater and greater numbers of patients are being maintained on either invasive or noninvasive nocturnal mechanical ventilation to rest ventilatory muscles.[11–14] The maintenance of adequate gas exchange is usually not the primary goal of therapy in this group of patients. A nasal mask[13,14] or negative-pressure apparatus[12] is the most common method of application. Because most of these subjects function for 16 to 20 hours per day without ventilatory support and many are not assisted on a daily basis, no indication for continuous monitoring exists other than for alarms identifying equipment malfunction.

The same can be said for patients with obstructive sleep apnea who use nasal CPAP nocturnally.[15,16] These subjects can function without the continuous use of CPAP and, although they may desaturate during sleep without the nasal CPAP, no rational indication exists for continuous monitoring of oxygenation. More appropriate are alarms on CPAP systems designed to notify users that the level of CPAP has dropped below a threshold for some predetermined period of time or that the equipment has failed. It is crucial that these system alarms not sound after momentary drops in pressure because pressure fluctuations are common with noninvasive CPAP systems. Periodic assessment of the efficiency of the CPAP system can be performed in the home, and data can be gathered periodically during sleep.[17]

Essential Positive-Pressure Therapy

Subjects in this group all *require* mechanical ventilatory support. It is likely that in these subjects, mechanical ventilation was begun after an acute event, and weaning was subsequently found to be impossible, mandating chronic ventilatory support. The majority of the patients in this group have tracheostomies, although some centers[18,19] maintain nearly continuous ventilatory support with noninvasive positive-[18] and negative-pressure[19] techniques.

Specific Needs of Adolescents and Adults. Criteria for discharge to the home for this group of patients

has been defined to include:[20] (1) absence of sustained dyspnea or frequent episodes of severe dyspnea and tachypnea; (2) acceptable arterial blood gases with an $F_{IO_2} \leqslant 0.40$; (3) absence of life-threatening cardiac dysfunction or arrhythmias; (4) manageable pulmonary secretions; (5) protected airway; (6) absence of significant nutritional deficit; and (7) psychologic stability—in other words, stable pulmonary disease or ventilatory dysfunction that is not rapidly progressing. Members of this group of patients rarely can be weaned from ventilatory support once it is established, but normally are capable of communicating to caregivers change in their overall status. Therefore, even in those patients requiring continuous 24-hour-a-day support, no indication exists for the routine use of continuous monitoring of gas exchange. Because these patients are medically stable, no change in gas exchange should occur without clinical signs of deterioration. However, it is essential to assure that ventilatory-assist equipment is functioning properly and is equipped with properly functioning and properly set alarms. Remote ventilator alarms and provision for calling an attendant should be provided. Recommendations for the use of continuous monitoring of gas exchange are not available. Neither the ACCP[20] nor monographs on home mechanical ventilation[21,22] recommend the use of these monitors. None of the persons that we have discharged from Massachusetts General Hospital on ventilatory support have required continuous monitoring of gas exchange in spite of the need in some cases for 24-hour-per-day support. If pulse oximetry or et_{CO_2} monitoring is included for this group, how would caregivers be trained to respond to the momentary changes that occur periodically? Would it be appropriate for the caregiver to notify the physician with each alarm or should the caregivers be trained to modify ventilator settings based on alterations in monitored variables? If we are not willing to recommend either of these alternatives, what purpose can the incorporation of these monitors serve other than to increase the level of anxiety?

During the periodic monitoring of the patient-ventilator system by respiratory care professionals, a number of assessments can easily be made (Table 3). These primarily include the "low-tech" monitoring techniques described by MacIntyre[23] and the assessment of compliance and resistance and work of breathing.[24] These values can be easily and rapidly determined during a home care visit and can provide useful information to trend status changes and identify early development of acute pulmonary problems. In addition to clinical assessment, spot checks with pulse oximeters and $etCO_2$ monitors can be performed. The problems associated with the accuracy and reliability of these measurements notwithstanding,[25,26] an argument can be made for periodic assessment of S_{pO_2} and $etCO_2$, provided baseline data correlated to arterial blood gases have been established and cardiovascular function is stable. The periodic tracking of S_{pO_2} and $etCO_2$ provides useful data for assessing efficiency of gas exchange, modifying ventilator-oxygen prescriptions, and identifying potential changes in status. This also ensures that a health care professional capable of evaluating the reliability of the oximeter and capnometer is the one using the instrument.

Specific Needs of Infants and Children. Members of this group differ somewhat from adults in their potential need for continuous monitoring because: (1) as a result of growth and development, their ventilatory-oxygenation needs change; (2) they have uncuffed tracheostomy tubes; (3) some can be weaned from ventilatory support in the home; and (4) with small infants, continuous flow, pressure-limited modes of ventilation are used in the home. As a result, greater controversy over the use of continuous monitors exists.

Table 3. Monitoring of Positive-Pressure Breathing Therapy for Adults and Adolescents in the Home

Elective Ventilation, CPAP, and IPPB
Monitoring of equipment function
Ventilation of the Ventilator-Dependent Patient
Monitoring of equipment function
Periodic monitoring
by pulse oximeter and end-tidal CO_2 analyzer*
by chest assessment
of vital signs and ventilatory pattern
of sputum production and consistency
of static compliance and system resistance
of airway pressure changes and work of breathing
of general clinical presentation

*No indication for continuous monitoring of S_{aO_2} or et_{CO_2}.

Many authors do not mention the need for additional monitors,[27,28] whereas others indicate the need for either apnea,[20,29] gas exchange,[20] or exhaled tidal volume monitoring.[20] It has been my experience that the need for continuous monitors of gas exchange is rare, but it may exist in some of the smaller infants.

The most convincing argument for the use of a continuous monitor relates to the limitations of the equipment used to ventilate infants. To my knowledge, no ventilator specifically designed for home use is available. Consequently, either ICU neonatal ventilators or adult home care ventilators are used. None of these units incorporates an exhaled volume monitor, and the intrinsic disconnect alarm can be easily 'fooled' because of the presence of a continuous gas flow. Thus, if a disconnect occurs and the tracheostomy tube adapter falls to the bed sheets, pressure may still rise during a positive-pressure breath so that the disconnect goes unnoticed. In addition, because of the presence of uncuffed endotracheal tubes, position changes can result in large variations in the volume of gas delivered. Although this can be compensated for by adjusting the ventilator settings,[30] the importance of positional leaks varies continually with the growth and development of the child. Many of these infants are also being actively weaned from ventilatory support. Because infants and children may not effectively communicate changes in their levels of cardiopulmonary stress as adults do, an objective monitor of physiologic function greatly increases safety during these weaning periods.

It is thus easy to argue for the use of either continuous oximetry, et_{CO_2}, or impedance apnea monitoring in the infant. In those children who require continuous ventilation because of central nervous system dysfunction in whom oxygenation is not an issue but in whom disruption of ventilation may be disastrous, impedance apnea or et_{CO_2} monitoring as a disconnect alarm may be indicated; whereas, in those infants in whom oxygenation is the primary concern (eg, those with bronchopulmonary dysplasia) pulse oximetry may be indicated. As smaller and sicker infants are sent home with mechanical ventilatory support, it is likely that the need for a continuous physiologic monitor will increase. Today, some rare situations do exist in which continuous monitoring is indicated.

All infants and children maintained on home mechanical ventilation should receive a detailed clinical assessment and oximetry and et_{CO_2} spot checks when the respiratory care practitioner visits (Table 4). The data obtained by these periodic evaluations are useful in assessing the magnitude of tracheostomy tube leak, assessing the need for adjusting level of ventilation and oxygenation, and evaluating the possibilities of weaning.

Table 4. Monitoring of Continuous Ventilation of Infants and Children in the Home

Continuous Monitoring
by pulse oximeter*—during oxygenation deficit, weaning, rapid growth change, tracheostomy tube leak
by end-tidal CO_2 analyzer* as disconnect alarm in small infants with CNS dysfunction
by impedance pneumonography*—in small infants with CNS dysfunction
Periodic Monitoring
by pulse oximeter and end-tidal CO_2 analyzer
by chest assessment
of sputum production and consistency
of airway pressure changes and work of breathing
of vital signs and ventilatory pattern
general clinical presentation

*Indicated only in the rare patient.

Bronchial Hygiene Therapy

I include under this heading aerosol therapy by small-volume nebulizer and metered dose inhaler, chest physical therapy, exercise programs, intermittent positive-pressure breathing treatments, and aerosol therapy by large-volume nebulizer. None of these techniques performed in the home requires the use of continuous monitors. Patients receiving this therapy do require periodic assessment, normally performed in the physician's office.

Sleep and Apnea Screening and Monitoring

Sudden infant death syndrome (SIDS) has been documented since the early 1800s.[31] The use of impedance apnea monitoring during sleep in the group

at risk has resulted in a significant reduction in mortality[32] and has been used for approximately 15 years. Any child suspected to be at risk for SIDS should receive apnea monitoring.

The development of techniques to screen neurologic and sleep disorders in the home has occurred rapidly. These techniques are used for screening prior to scheduling formal sleep laboratory evaluations[33,34] and frequently provide detailed diagnostic information that can be used to guide existing therapy.[17] Some of the available equipment allows the monitoring of four to eight channels of physiologic data[35] including heart rate; respiratory rate; chest impedance; S_{pO_2}; nasal and oral airflow; and electroencephalographic, electromyographic, and electro-oculographic activity. This facet of home care diagnostics (while in a developmental stage) appears to be a necessary and appropriate extension of monitoring, especially in light of the difficulties encountered in scheduling patients for sleep studies in laboratories that frequently have a backlog.

In Conclusion

The subject of continuous and periodic monitoring in the home is filled with controversy, but lacks data or guidelines to clearly establish indications. However, because large numbers of sicker, technology-dependent patients are being sent home, monitoring applications can be expected to further expand and develop. Respiratory care would greatly benefit from a consensus conference to clearly define the Whos, Hows, and Whens of patient monitoring in the home.

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Kacmarek Discussion

Martin: I think it is appropriate to send some babies home on supplemental oxygen. Weaning these babies is difficult and I think pulse oximetry clearly is valuable and effective in a clinic setting. It's known that these babies don't improve very rapidly, and I see no reason why they can't come in once a week or every other week, have their pulse oximetry done, and have the appropriate adjustments in inspired oxygen made at that time. I don't think capnography is very helpful in weaning these babies because they often have a high P_{CO_2} (60-80 torr) with normal pH (compensated respiratory acidosis). Sometimes there is tremendous pressure exerted on us to discharge ventilator-dependent babies, but most centers that I am aware of have step-down arrangements with smaller hospitals where these babies can be mechanically ventilated. To ventilate them at home and attempt to simulate an intensive care unit in the home is fraught with problems.

Kacmarek: When you send children home on oxygen therapy, you perform oximetry 'spot checks' when they come in for visits, right? You're not sending them home with an oximeter, are you?

Martin: No, the only equipment we're sending them home with is for oxygen administration (and cardiorespiratory monitoring).

Kacmarek: What types of monitors—other than ventilator alarms—do you use for the children that you do send home on mechanical ventilation for whatever reason?

Martin: Rob can correct me if I'm wrong, but we don't do that.

Kacmarek: You don't send patients home on mechanical ventilators or . . . ?

Martin: No.

Chatburn: Very rarely, the occasions that I recall in which we've sent kids home on mechanical ventilators have been with much older bronchopulmonary dysplasia (BPD) kids who just needed nocturnal mechanical ventilation—and in these cases we have not used monitors other than those that were supplied with the ventilator.

Kacmarek: Neither have we. However, if you talk to other home care providers around the country, there are a fair number of children—although I don't think it's appropriate—who are being sent home with sophisticated ICU ventilators (on occasion), with pulse oximetry, and in some cases end-tidal CO_2 monitoring to be maintained in the home care setting.

Chatburn: I think you made the statement that trying to set up an intensive care unit in the home environment is probably not a good idea, and it sounds like what you're saying is that some places are needing all these monitors because they have an intensive care-like environment. I had one experience in trying to do that with a small child who had Ondine's curse and who needed intensive monitoring. What inevitably happened was that there were so many false alarms that eventually for one reason or another the alarms didn't get turned on, with a fatal consequence.

Kacmarek: I agree. I guess what I'm saying is that there needs to be a consensus about home monitoring of oxygen and mechanical ventilation that doesn't exist today. Home care providers get calls to move patients out of the hospital in a day or two with an ICU ventilator and to utilize extensive noninvasive monitoring in the home setting. You and I may not be doing this, but I don't think that is true throughout the country. There are absolutely no standards, no guidelines, no data whatsoever to guide the application of respiratory care in the

home setting—other than reimbursement guidelines.

Giordano: I'd like to qualify your statement about the reimbursement in Texas and California under Medicaid. That reimbursement only applies to the professional component of respiratory care for support of a chronic ventilator-dependent patient. The definition of that chronicity goes back to the ACCP's task force: 6 hours/day for 30 days. The more conventional forms of respiratory care provided by the respiratory therapist are still not recognized for reimbursement. I'd also like to add that the quandary that the government is in, given conversations I've had with representatives of FDA and HCFA, with regard to monitoring (specifically pulse oximetry) is that they are not aware of any pulse oximeters that have been certified for home use. So the government on one side (FDA) is saying that you can't use them in the home; therefore, the government (HCFA) cannot pay you to do so. So, it's more than just money here. We must have equipment that is suitable for home use, and it must be brought in 'through the front door.' We know it's being brought in 'through the back door' and so do the regulators. Because there is 'no other game in town,' they're winking at the regulations right now, but they're not willing to pay for it.

Kacmarek: Some critical standards need to be developed for noninvasive home monitoring, specifying the types of patients for whom it is appropriate to provide more than the monitoring systems that exist on mechanical ventilators. For example, if you have a patient who only requires mechanical ventilation 12 or 14 hours a day, clearly he can survive for short periods of time off the ventilator if he becomes disconnected. I'm not sure a good argument can be made for the use of an expensive monitor in the home for this type of patient.

Mathews: The draft documents from the FDA for apnea monitor standards did not contain any discussion of who should be monitored or why monitoring should be done. These FDA standards only addressed the how. We need to address the who and why in the standards somehow. The question becomes, Should it be the FDA's responsibility to determine which types of patients should receive noninvasive monitoring in the home?

Ritz: I appreciate the problems related to reimbursement. What I see is that we treat children in the home setting a little bit different than we treat adults. At my institution, we have found that in the management of adult ventilator-dependent patients (particularly those with high cervical injuries who have no pulmonary function) the alarms provided with the ventilators that we've used are inadequate. And, not to air my dirty laundry, but my institution is one of many (unfortunately) that has had a catastrophic occurence because ventilator-disconnect alarms have not proven to be fail-safe, particularly in that patient population. What we've gone to—rather than monitoring to detect ventilator disconnect—is monitoring whether the patient is breathing; and we do this with an impedance apnea monitor. We probably do a little bit of overkill because we've been scared—we use a combination impedance apnea monitor and oximeter—but I could make a case for saying that ventilator-dependent adults in the home setting be managed in that fashion. In a straw poll that we took, everybody who managed high-cervical-injury, ventilator-dependent patients with no pulmonary functions at one time or another has had an unfortunate incident occur.

Kacmarek: I'm not saying that doesn't happen in the hospital setting, but those patients who are sent home are usually relatively stable. Dr Back and his group from New Jersey maintain patients with virtually no tidal volume on noninvasive approaches to mechanical ventilation (24 hours a day!).[1,2] There are techniques (eg, frog breathing) that patients can be taught to use during periods when they might be disconnected from the ventilator—any monitor can fail. I don't think it's realistic for us to think that we can create a totally fail-safe environment—that we can cover every conceivable detrimental thing that might occur. I think we need to decide what is affordable, what is really necessary, and what's going to increase the likelihood of our being able to maintain patients at home. You know every time you go to that higher level of monitoring, it simply intensifies the anxiety experienced by the family and/or caregivers. Even many of the mechanical ventilators that are used in the home, as far as I'm concerned, are inappropriate because of the number of monitors they possess—some of them have 12 monitors that are going to alarm, that are going to create the situation described by Rob (Chatburn). I think this is a critical problem! We all see it in the ICU—alarms ignored, alarms shut off. It is not reasonable to expect that this won't also happen in the home care setting.

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2. Back JR, Alba AS, Lee M, Rideau Y. Long term respiratory rehabilitation in the treatment of neuromuscular disease. Ann Readapt Med Phys 1983;26:101-109.

Krieger: I'd like to extend that thought—If you use a monitor that doesn't detect what it's supposed to detect, is it better to have no monitor at all? For example, if you have an obstructive apnea patient being monitored with an impedance device and the apnea is not detected, is it better not to monitor at all? The patients who I have on nocturnal ventilation in the home are not on any monitors even though I'd love to put them on lots of things.

Welch: I'm particularly struck by the lack of data in the field of home health care, and I wonder if mortality and morbidity rates might be available from insurance carriers. If an incident occurs on a ventilator-dependent patient in the home, the insurance carriers should have this data, if anybody should. Are they open to this kind of information sharing?

Kacmarek: I don't know. I've never looked into that area to secure data.

Giordano: I've tried to approach both the insurance association and individual carriers; they won't provide us any data whatsoever—least of all anything about morbidity and mortality! We get more from the federal government than we get from any of the private carriers. I would also like to put a couple of things into perspective: The issue of monitoring within the context of respiratory care in the home is unusual! I think that a lot of people are advocating home mechanical monitoring just so their consciences will be assuaged when they send people home—they think that the only thing that keeps people in the hospital is the fact that mechanical monitoring is available there. This reasoning completely overlooks the fact that skilled, educated practitioners are also available to patients while they are in the hospital! These *human* resources don't generally exist if you look across the care spectrum to the home, or even to the SNFs, where we see an increasing number of ventilator patients going.

Pierson: (That means "skilled nursing facilities"—nursing homes, guys.)

Giordano: Right, well, I'll stick close to the editors on that. To bring things into perspective: In 1987, three quarters of a billion dollars (ie, $750 million) reimbursement was applied for—just to HCFA—for respiratory care equipment. Of that, $622 million was paid (a very small fraction of that amount was related to ventilators) and that was spread over about 391,000 Medicare beneficiaries.[1,2] Oxygen is the key thing here! It is our biggest concern; it touches more patients than anything else; and that's where the abuses occur (eg, patients not getting what the physicians have ordered or receiving equipment that does not work) because there are no skilled hands, there are no educated people; and it's left to family caregivers and the like! Bob (Kacmarek), do you have other criteria that deal with the psychosocial needs (ie, the network that needs to be in place to support the patient if he meets the physiologic criteria to be sent home)?

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2. American Association for Respiratory Care, Food and Drug Administration, Health Resources and Services Administration. Final report of the consensus meeting on home respiratory care equipment. Dallas TX: AARC, 1989.

Kacmarek: Yes, whenever we send a patient home, we review the home setting in terms of the physical layout and the educational or learning capability of the caregivers. It's rare though that highly skilled caregivers are required to assist the family in the home. Most of the time if you have a family situation—a single parent taking care of a child or an elderly person being cared for by a wife or other family member—you usually can get away with unskilled caregivers being trained to do the minimal tasks that are necessary because in many cases the patient simply needs somebody to watch him, somebody who will be available to lift, bathe, and feed him. We get into a problem with reimbursement because too many centers attempt to send patients home regardless of what the family situation is, with RNs as caregivers 24 hours a day. We recently had a big battle in which we were fortunately able to convince everybody that a patient with ALS (who had about 6 months to live) didn't need to go to a rehab center and didn't need nurses around the clock, but simply needed a caregiver for 12 hours a day to sit at the bedside during the night while the family members had a little free time and could sleep without worrying about the patient. It would have ended up costing a fortune to maintain the patient at home if 24-hour RN coverage had been employed! I think it's critically important to evaluate every aspect of a patient's environment—the capability of the caregivers to provide adequate support, as well as the adequacy of reimbursement to cover the home care services that are going to be provided.

Resource Management for Noninvasive Monitoring

Ray Ritz BA RRT

Introduction

Noninvasive monitors seem innocuous enough at first glance, but they all have two things in common: They cost money, and they require people to operate and observe them. They come in all shapes and sizes, and supply an ever-widening array of physiologic data. They can be easily and quickly connected to the patient without carrying the risk of infection or injury that invasive monitors do. Their manufacturers strive to give them the appearance of simple operation and reliable, accurate performance. For some patients, the use of these monitors has become a virtual community standard. For example, noninvasive monitoring of arterial oxygen saturation is considered by many to be the 'fifth vital sign.'[1] It is difficult to "just say no" to the possible contribution of such monitors to the process of providing health care.

Overutilization of these tools can cause a variety of problems. Clinicians may become dependent on these systems and less aware of the patient. Each monitor requires some attention from staff, and the cumulative result of increasing numbers of monitors is increased activity (work) for the staff. Also, these devices can be costly to operate and maintain.

I believe that many noninvasive monitors have clearly been shown to be helpful in managing today's critically ill patients. The health care industry is eager to use any device to reduce the risk of litigation and to meet society's expectations of high quality. Noninvasive monitors are necessary, and they are here to stay. They will continue to evolve; today's challenge is to improve our management of them at the same pace. We can optimally use or misuse human and financial resources by the decisions we make on noninvasive monitors. How many of which ones? is a question that can be adequately answered only by careful analysis of each institution's needs.

Determining Your Need

The need for any type of monitor is based on the institution's size and patient mix. The hospital with a large neonatal intensive care unit may require more transcutaneous monitors than an institution of similar size that treats primarily adult patients. Indirect calorimetry may be required at a hospital that operates an intensive care unit (ICU) for burn patients but not at a small institution with a limited number of critically ill patients.

Noninvasive monitors can be classified as continuous or intermittent, routine or therapeutic.[2] For example, capnographs can be used either continuously 24 hours a day or for short intervals to monitor tolerance to a physiologic challenge. A pulse oximeter may be used routinely to monitor all mechanically ventilated patients, but an indirect calorimeter may be used only on selected patients whose nutritional status is compromised and whose support must be carefully adjusted.

The cost-benefit ratio of a monitoring system is based on the initial purchase price, operating costs, and maintenance expenses compared to reductions in labor intensity, conservation of other resources, and improved quality. This is not a straightforward calculation. Assigning a dollar value to changes in quality or accurately determining time and resources saved is difficult at best.

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Noninvasive Monitors

Capnographs

Capnographs cost from $3,000 to $10,000 depending on the model. They are available as stand-alone devices or combined with pulse oximeters as a single unit. The major drawback to capnographs from my viewpoint is the need for frequent attention during continuous use. Moisture and debris in the patient's exhaled gas tend to foul the gas sampling line or analysis chamber, making the device inaccurate or nonfunctional. A variety of filtering methods are used, but these too may need attention. Capnographs can be annoying to calibrate and usually require special gases for this purpose, which adds to the operational cost. The requirement for frequent assessment and manipulation makes this device moderately labor intensive.

Capnographs can be useful for determining physiologic dead space and carbon dioxide production. Patients at risk for respiratory failure during weaning may benefit from capnographic monitoring, but most patients can be weaned from mechanical ventilation without it. Patients at risk for hypoventilation, such as those experiencing sleep apnea or those receiving epidural narcotics, can be monitored with capnographs, but other methods (RIP or chest-wall impedance devices) may be easier to use. Some unique applications should also be considered. Nontraditional modes of ventilation such as the use of inverse inspiratory-expiratory (I-E) time ratios may be optimized more easily by continuously monitoring end-tidal CO_2.

The Society of Critical Care Medicine's "Task Force on Guidelines"[3] has stated that the capability of monitoring ventilation is desirable for some ICU patients. Utilization criteria should target patients whose ability to maintain adequate ventilation is suspect. The number of capnograph units required should be determined by the institution's size and patient mix. A rule of thumb may be to purchase one capnograph for every 100-200 hospital beds.

The simple CO_2 detector can be used to detect apnea. Less expensive than quantitative devices (about $2,000), these detectors use the same infrared absorption principle but give no data on the level of CO_2 sensed. When CO_2 is not sensed intermittently, apnea is assumed and the alarm sounds. They are simple to use and can replace the more expensive capnograph for some applications.

It has been suggested that capnographs may be useful in verification of endotracheal tube placement after intubation.[4] Although a setting such as the operating room may be well suited for this application, unstructured emergency situations in other areas may not be. The time required to deliver the monitor and connect it to the patient may preclude its use.

A small, nonmechanical, single-patient-use device is now available that uses a color-change indicator (like litmus paper) to sense the presence of exhaled CO_2. It can be easily stored with intubation supplies and used when tube placement is in question. At about $15 each, the per unit cost may dictate selective utilization.

Cardiac Output Monitors

Noninvasive cardiac output monitors are slowly gaining a foothold in today's array of accepted methodologies. These monitors have the advantage of being able to quickly quantify a sometimes critical physiologic variable in patients with questionable hemodynamic status. In an ICU or intermediate care unit (IMCU), this application is attractive. The danger lies in potential overuse. The criteria for monitoring cardiovascular function should include unstable myocardial infarctions, ARDS, or the application of PEEP.[5] This somewhat complex device requires a skilled user to ensure its accurate operation, and it is expensive (about $20,000).

The interpretation of data from Swan-Ganz catheters is still complicated even with our current body of experience.[6] Although the noninvasive determination of cardiac output is a 'neat trick,' the data provided on pulmonary artery pressure, left ventricular pressure, and total thoracic fluid volume, are expressed in bioimpedance terms—requiring the learning of a whole new set of concepts to make correct therapeutic decisions. Noninvasive monitoring of cardiac output is promising, but its new jargon combined with the need for additional studies of its accuracy and limitations make it slow to be accepted.

There is little reason to monitor cardiac output outside of ICUs and IMCUs because patients needing this monitoring also require more frequent attention than that available on general duty floors. Due to our limited experience with noninvasive cardiac

output monitoring, it is difficult to make a recommendation on the number of devices an institution may need.

Impedance Apnea Monitors

Impedance apnea monitors are simple to use and relatively inexpensive ($1,500-$4,000). Some models couple an apnea monitor with a pulse oximeter to provide respiratory rate, oxygen saturation, and heart rate data—a combination that may be well suited to some patients at risk for apneic periods. Patients must be carefully screened to ensure that those with obstructive apnea are not monitored in this fashion alone because chest-wall movement may continue in spite of the absence of ventilation.

Impedance apnea monitoring is well suited for most patient care areas as long as certain precautions are taken. In areas where staff attendance is assured (ICU and IMCU), the device's audible alarm is generally sufficient. On floors where the patient-nurse ratio is high, arrangements must be made to provide remote alarms that can be heard by staff who are caring for other patients on the floor.

Patients receiving epidural narcotics can be safely managed with impedance apnea monitors, although not all will require them. Patients of advanced age, those with severe COPD, or those recovering from extensive surgery are at greater risk. Most patients managed with epidural narcotics need only hourly assessment of vital signs to monitor tolerance of the treatment.

The number of stand-alone impedance apnea monitors needed in each institution will depend on the number of patients cared for who fit the above categories. I believe that 1 to 3 units/300 beds is sufficient unless the institution offers services that increase the number of special needs. In ICU areas, today's cardiac monitors often have the ability to do impedance chest-wall monitoring, which can reduce the number of stand-alone units needed.

Metabolic Carts

Indirect calorimetry has been available for years as part of the diagnostic services offered by pulmonary function laboratories. Only recently has it become feasible to provide such monitoring at the bedside. Metabolic carts are delicate, complex systems that must function with a high degree of accuracy. The manufacturing process that enables them to perform adequately in the demanding and sometimes abusive bedside environment is just now being perfected. At a cost of $20,000-$60,000, metabolic carts are among the most expensive noninvasive monitors to purchase. At the same time, some controversy persists regarding need for and benefit from such monitoring.

The complex metabolic cart requires a skilled operator to consistently obtain accurate measurements. Manufacturers have attempted to make these devices as intuitive as possible, yet numerous factors complicate their use. The nature of the resting energy expenditure determination dictates that measurements be made only after stability is reached[7]—with patients fasting for 2 hours and resting supine for 30 minutes before the test, with the test environment quiet and serene. Only the close observation and control of a skilled clinician can eliminate operator error and discard results obtained under unstable conditions.

It may be premature to make a recommendation at this time about the number of metabolic carts needed by an institution. When the procedure is carefully performed, indirect calorimetry can provide accurate assessment of a patient's nutritional needs.[8] However, results of a survey reported in 1988[9] suggest that of the 60 indirect calorimeter users reporting, more than half believe that values produced by their units are not significantly different from calculated, or predicted, values. Two thirds of those users performed fewer than 10 tests per month. I believe that this clearly reflects a lack of established protocol and criteria for utilization.

I believe that an institution contemplating the purchase of a metabolic cart would be wise to start by organizing a committee charged with designing reasonable protocols. This group should consist of the physicians who will order the studies, dietitians, respiratory care practitioners, pharmacists, and nurses. Regardless of how simple the study, each patient will need monitoring for a minimum of 30 to 60 minutes. Frequent attention by the operator is required to ensure correct functioning of the device and to control the patient environment. This makes indirect calorimetry labor-intensive.

With criteria for selecting patients established and a reasonable protocol for testing agreed upon, most hospitals of moderate size will not need more than

one metabolic cart. Institutions with high volume or specialized services (such as burn treatment, frequent major surgeries or trauma, or treatment of other complex and serious illness) may require two. Without the controls provided by an organized system, a metabolic cart can be an example of institutional overconsumption and unnecessary technology.

Bedside Pulmonary Function Screeners

Computer technology has helped bedside pulmonary function screening devices achieve a sophisticated yet simple level of operation. A wide range of spirometric values can be measured easily regardless of the patient's location. Some units are hand-held; others can be conveniently moved about on a small cart. Prices range from $1,000-$10,000 depending on the model.

Preoperative screening can be useful in patients with lung disease and in those facing thoracic surgery, but is not necessary for all surgical patients.

Evaluating a patient's response to bronchodilator therapy is simple. The limitation to providing this service may be the sheer number of patients who are receiving aerosolized medications. The respiratory care department in an average hospital may need one table-top unit and several hand-held models for every 300 beds.

ICUs have little need for sophisticated spirometry. Determination of lung volumes of mechanically ventilated patients would be useful in some conditions, but is not now technically feasible except for purposes of research. The measurements of interest in mechanically ventilated patients are spontaneous rates and tidal volumes and indicators of strength and reserve. These can be gathered with inexpensive, hand-held devices by the clinician at the bedside. This approach is desirable because the clinician's observations during the tests are as important as the numbers gathered.

Pulse Oximeters

Pulse oximeters—perhaps the simplest and easiest noninvasive monitor to use—can provide valuable information about the availability of oxygen to the tissues, a critical aspect of physiology. In our institution—and probably at yours also—it has become the most frequently requested noninvasive monitor. At the same time, it is becoming an excellent example of a technology in need of direction. Although community standards continue to develop, it has become clear that continuous pulse oximetry should be available to any critically ill ICU patient in whom the adequacy of oxygenation is in question.[3] ICU patients who have maintained a stable, normal oxygenation probably do not need it at all. The number of devices required for the critical care areas of the hospital is approximately one for every 1-3 ICU beds. Institutional convention and resources will dictate the exact number.

Hospitals with IMCUs or noninvasive monitoring units (NIMUs) will need a similar number of continuous oximeters for those patients. Perhaps it is the patients who are cared for on the regular nursing units, the 'wards,' who pose the most complex problem. Continuous monitors operating with only intermittent observation seem to be wasted. Patients who need close observation should be moved to nursing units where staff are available. An occasional ward patient may have need for continuous oximetry, but that should be the exception—not the rule.

Single saturation measurements, or 'spot' checks, on miscellaneous patients throughout an institution can present a major difficulty for a respiratory care department. Even though a single spot check takes only 5 minutes to perform, 25 will take more than 2 hours. Excessive ordering of oximetry checks can cripple a department. At my institution, Harborview Medical Center (HMC) in Seattle, Washington, a quality assurance monitor revealed that in 88 individual non-ICU spot checks, only 3 pulse oximeter saturations (S_{pO_2}) were found to be below 90%. Those three were measured on patients with diagnosed COPD whose S_{pO_2} was chronically below the 90% threshold but who otherwise appeared stable. For those same 88 ordered measurements, no comment was made in the patient's chart about the S_{pO_2} by either the physician or the nurse and no change was ordered in oxygen therapy.

In response to a 'plague' of requests for spot-check oximetry, the staff at HMC have developed—in jest—two screening tests to detect clinically significant episodes of desaturation. These are termed the "arm-lift saturation test" and the "two-ring saturation test."

If patients can lift their arms and extend their fingers on request, their saturation is probably $\geq$ 94%. The other approach is to call the patient's room. If the patient answers the phone in two rings, again we assume that oxygen saturation is $\geq$ 94%.

Some respiratory care departments have responded to the overutilization of spot checks by transferring responsibility for performing them to the nursing staff and ordering physicians. This may solve the problem for the respiratory care staff but not for the nurses and physicians—or the patient. Responsible, criteria-based ordering is the solution for everyone.

Some respiratory care departments may not want to give up their 'turf,' and there are logical reasons for respiratory care to retain control of oximetry. Respiratory care practitioners have specialized training in the use of sophisticated technology and the assessment of pulmonary physiology that makes it easier for them to determine whether the oximetry data matches the clinical situation. Episodes of CO_2 retention and pH abnormality may go undetected if clinicians rely solely on pulse oximeters to assess blood-gas status. When a patient manifests symptoms of acute respiratory failure, an arterial blood gas is indicated. Either through aggressive education and quality assurance or by retaining control of oximetry, respiratory care departments can be instrumental in assuring appropriate utilization and optimal patient assessment.

Another common problem with pulse oximetry is that this technology does not function well in patients experiencing poor peripheral perfusion—the patients for whom oxygenation monitoring is most useful.

The last consideration regarding the management of pulse oximetry is the control of operational costs. A great variety of probe styles are available. Reusable probes would seem to be cost-effective, but are expensive to replace when broken. Maintaining proper placement of reusable probes on some patients can be difficult; disposable probes are easily connected and maintained on the patient, but they are expensive. A reasonable approach may be to do as much short-term oximetry ($\leq$ 48 hours) as possible with reusable probes and reserve the disposable probes for long-term monitoring. Disposable devices may be well-suited to situations in which the probes and cables are subjected to greater than normal abuse (as in agitated or uncooperative patients).

Debate continues over what constitutes the best and most accurate pulse oximeter. Most models appear to function adequately under normal conditions. Choices should be based on a fair clinical trial, competitive price, durability, and service.

Transcutaneous Monitors

Transcutaneous monitors provide data that relate to the oxygen and carbon dioxide content of capillary blood and its pH. Although not direct measures of arterial blood gas values, transcutaneous values (P_{tcO_2} and P_{tcCO_2}) can be closely correlated to and trend well with arterial changes and are particularly valuable in neonatal and pediatric applications.

The correlation between transcutaneous and arterial values is not as close in adults as in children. Also, changes in peripheral blood flow at the sensor site may affect values and fail to provide data representative of the adult's overall physiology.

The higher purchase (from $5,000 to more than $10,000/unit) and operating costs of transcutaneous monitors compared to the costs of oximetry and capnography have contributed to the limited utilization of transcutaneous monitors in adults. Probes (miniaturized Clark or Severinghaus electrodes) are delicate and expensive. Also, the probe site must be changed every 2-4 hours to prevent burning the patient. Calibration of the units requires special gas mixtures, and electrode membranes must be replaced periodically.

As the technology improves, these devices may become more economical and simpler to use. Devices incorporating transcutaneous CO_2 electrodes and pulse oximeters are available. It is difficult to make recommendations on the appropriate number of transcutaneous monitors for an institution that does not care for pediatric or neonatal patients. One or two may be useful for institutions with large numbers of critically ill patients who require mechanical ventilation, but adequate monitoring can be accomplished with other devices. Institutions caring for pediatric and neonatal populations may need one P_{tcO_2}-P_{tcCO_2} monitor for every 2-3 mechanically ventilated patients. Spontaneously breathing, critically ill patients with unstable pulmonary physiology may also be candidates for such monitoring.

Ventilation Monitors

Ventilation monitors are one of the newer arrivals on the noninvasive monitoring scene. For years,

researchers have been able to monitor and record any number of parameters related to the mechanics of ventilation using computers and sophisticated pressure transducers and pneumotachographs. The current generation of ventilators can provide similar data, and ventilator manufacturers are giving us the ability to display and manipulate it. Depending on the system used, pressure, flow, and volume waveforms can be examined and combined. Compliance, inspiratory-expiratory time ratios, airway resistance, inspiratory effort, and other measurements can be made by various ventilation monitors. The problem with these sophisticated monitoring systems is that they cannot always provide the data necessary for subtle judgment calls. We have found that simple things like counting the patient's respiratory rate are difficult for some ventilator monitoring systems, and values for compliance and inspiratory effort may differ from those obtained manually by the clinician.[10]

Some ventilators (eg, the Bear 5) incorporate components to provide graphic and digital data output as is. Others require a communication package (cost $\geqslant$ \$1,000), and then the ventilator must be connected to a computer system with display (cost $\geqslant$ \$3,000) and loaded with the appropriate software (cost $\geqslant$ \$1,000). Once the system is configured, you must learn to interpret what you see. This data can be helpful in unique ventilation trials such as inverse-ratio ventilation or in a patient with severe airflow obstruction.

Given the general respiratory care practitioner's limited exposure to this type of monitoring, buying such a monitoring package for every ventilator may not be the best plan. The resolution and accuracy of these systems have not been clearly determined, and most mechanically ventilated patients do not need it. It seems realistic to assume that in the future respiratory care practitioners will use such monitoring regularly on difficult patients. As understanding of the benefit of the data provided by sophisticated ventilation monitors and confidence in their validity increases, utilization will increase.

Purchasing Plans

A department has several options when considering the purchase of any noninvasive monitor. A direct purchase is the most economical way of obtaining a costly device. Most institutions consider any instrument costing \$500-\$1,000 a capital item. Capital funds are usually separate from the monthly operational budget and are planned and budgeted at the beginning of each fiscal year. When capital dollars are limited or when the need for a device occurs unexpectedly, one may be forced to consider the use of operational dollars to acquire the device. The acquisition can then be accomplished by renting or leasing to own.

Renting-to-own is useful when the ultimate acceptance of the device is in question. With this option, a device is rented on a month-to-month basis and, after a designated period of time, the device becomes the property of the institution. If the rental is stopped before that point, ownership is retained by the vendor. Generally this is the most expensive method of purchase. The advantage is that the purchase can be terminated if it becomes apparent that the device is not useful to the institution. The renting-to-own option is generally not available for more expensive monitors.

Leasing can be less expensive than renting-to-own, the difference being that the institution is contractually bound to pay the monthly fee until the device is paid for. This option is attractive to those departments that do not have sufficient capital budgets to allow an outright purchase.

Calculation of Operating Costs

The operating costs of a noninvasive monitor can be attributed to four sources: disposable or consumable supplies, preventive maintenance costs, incidental repair cost, and labor costs associated with employee education and the time needed to operate and observe the device. A device such as a pulse oximeter may seem inexpensive to operate, but if an institution chooses to use disposable probes, the cost can become important. On the other hand, if the breakage rate in a specific department is high on reusable probes, disposables may help reduce costs. Each type of monitor will need supplies. Making an accurate estimate of usage and cost is essential when considering the addition of new technology.

One of the smartest things a manager can do is to ensure that adequate preventive maintenance is provided for equipment. Well-maintained devices save clinicians' time at the bedside and are necessary for patient safety. Preventive maintenance also helps avoid more costly repairs later.

Any device will break at some time—from normal wear and tear or from accidents. Patients may not perceive the need to be gentle with these sophisticated monitors. Putting down a bedrail can quickly and abruptly move a $5,000 device from the table to the floor. Items even break spontaneously, and a detailed investigation may reveal that no one cut the cable in half—it just 'happened' by itself. Keeping spare parts on hand and sending devices out for repair is costly. It may be necessary to rent a replacement when an instrument is sent out for repair—further increasing operating costs.

Estimating the purchase and operating costs can be complicated. Figure 1 compares the purchase cost

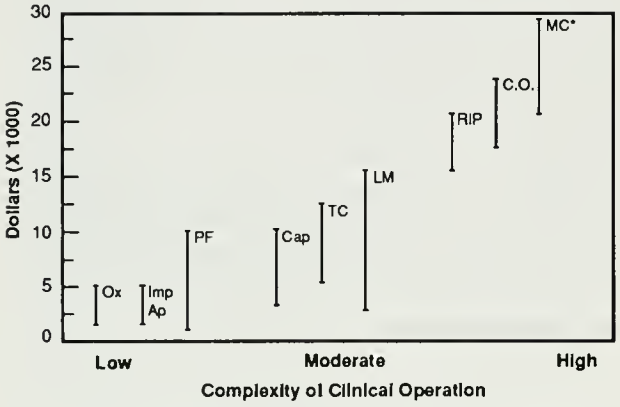

Fig. 1. The range of purchase costs (manufacturer's list price) of noninvasive monitors is plotted on the vertical axis and the overall complexity of each device on the horizontal. The complexity rating is based on information provided by users, manufacturers, and the author's personal experience in his department. The rating includes the physical setup and operation, the education or training required, and the complexity of the data output from each device. *Some models of metabolic carts cost as much as $60,000. (Ox = oximeters, Imp Ap = impedance apnea monitors, PF = pulmonary function screeners, Cap = capnographs, TC = transcutaneous monitors, LM = lung mechanics monitors, RIP = respiratory impedance plethysmographs, C.O. = cardiac output monitors, MC = metabolic carts.)

of the monitors discussed here against their overall complexity. Historical records or information from institutions with more experience may be the best way to accurately anticipate the fiscal impact of a new technology. Gathering information from sources other than the manufacturer is prudent because unanticipated problems and a less-biased perception of usefulness and durability of a device may be revealed.

The labor costs of noninvasive monitors are difficult to quantify—the more complex, the more costly; the more fragile or sensitive, the more costly. An indirect calorimeter—on the surface—may seem simple to operate, but ensuring that the bedside environment is conducive to performing the study may be very labor intensive. A device such as a bioimpedance cardiac-output device may be quickly applied to the patient, yet may require extensive educational effort to ensure adequate comprehension and utilization of the data produced. The more devices in use, the more points to be checked and observations to be recorded. More is not always better.

Resource Management

The solutions to problems of appropriate utilization of noninvasive monitors are varied and complex. Caution must be used both to avoid overutilization and to accomplish adequate monitoring as defined by community standards. The concept of community standards is ephemeral. Vendors will be quick to show managers every piece of evidence that alludes to the need for their device in as many situations as conceivable. Our goal should not be to have every device available for every patient—just because it exists.

We must also be careful not to try to turn every ICU or IMCU into an operating room (O.R.). Technical requirements for an O.R. will always exceed those of other areas of the hospital. Development of logical and reasonable criteria for utilization of new forms of technology is essential. These criteria should include the target population for each device and reasonably defined points at which to begin monitoring and to discontinue monitoring.

Physicians ultimately control the resources used to care for patients. Conservation strategies advocated by Birnbaum[11] (Table 1) include physician education as an integral component of the resource-management process. Although this strategy must be a part of any institution's plan for resource management, it has not been clearly shown to have a positive impact on cost containment.[12] Systems that allow a designated group of physicians to control utilization of respiratory care services have been shown to reduce inappropriate or unnecessary therapy.[13] An additional tactic that

may be helpful is maintaining a well-trained group of respiratory care practitioners who possess the skills to interact effectively with the ordering physicians. This method has also been useful in reducing the frequency of inappropriate orders for respiratory care services.[14]

Utilization review, observation of clinical practice, and quality assurance monitors all help pinpoint when utilization is appropriate and when it is not. I believe that almost any system devised by a department will need fine-tuning after it has been used for a while. An ongoing monitoring process is not only a mandate of the Joint Commission on Accreditation of Healthcare Organizations but also an important part of any safe resource-conservation process.

Noninvasive Monitoring Units

Another method for controlling and appropriately utilizing noninvasive monitors is the development of a specific unit in the hospital for that purpose. It has been shown that 24-40% of ICU admissions are to provide increased monitoring, not to provide active treatment.[15,16] Concentrating those patients who need closer observation in a designated area can help reduce the staff needed and still provide a safe environment. This is not a new idea. Monitor stations have been in existence for years in ICUs. One individual can observe numerous monitors freeing other staff to provide direct care to other patients.

The recommendation for the creation of noninvasive monitoring units to reduce the utilization of costly ICU beds seems appropriate. This setting in which the patient-nurse ratio is higher than in the ICUs yet lower than in the wards seems ideal to capitalize on the advantages of many noninvasive monitors. An institution intending to exercise this option may be well advised to plan the venture carefully.

To obtain maximum efficiency from this scheme, equipment that can be integrated into a comprehensive system should be purchased. A monitoring station, similar in design to that found in an ICU, improves efficiency and utilization of personnel. Allowing members of a well-trained group to apply a criteria-based protocol can be effective in assuring appropriate, economical utilization of a resource.[17,18]

Table 1. Strategies for Resource Conservation and Constraint*

Physician education
Physician participation in decision making
Quality assurance monitors
Utilization review
Observation of practice
Established criteria for equipment replacement
Minimization of the influx of new technology

*Adapted, with permission, from Reference 11.

Summary

Today we are faced with an ever-expanding array of technologies that allow monitoring to be accomplished more accurately and more easily than before. Each institution's needs differ—yet, the methods for determining appropriate utilization are similar. A rational method of utilization includes a criteria-based process for deciding who will be monitored and when. An accurate estimate of the number of units required, their purchase and operating cost, the complexity of operation, and the potential impact on both patients and staff must be calculated. An ongoing educational program for respiratory care practitioners, nurses, and physicians should be implemented to communicate the values and limitations of each device.

These technologies can be valuable adjuncts to our process of caring for patients. Used appropriately, noninvasive monitors can fulfill their promise of improving health care. However, they do carry the risk of increasing the overall cost of health care and of distracting us from personally assessing our patients. The existence of an expensive device is not reason enough to employ it. If it were, I would have a difficult time responding to my wife's frequent question of, "If they can send one man to the moon, why can't they send them all?"

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Ritz Discussion

Rodriguez: I gather that you don't support 'spot checks?'

Ritz: They're my least favorite, yes!

Rodriguez: I am aware that various departments feel that this is a good way to review the appropriate utilization of oxygen on the floors. What's your opinion?

Ritz: I guess what I'm down on is therapists doing spot checks because one of the things that really affects productivity is transit time from one area to another. Without even doing a time-motion study—just listening to my therapists (they go to the seventh floor to do a spot check, they go to the third floor, then they up to the fourth floor, and then they go down to the second floor)—I think if you want to provide spot checks, the only reasonable thing to do (given the ease of pulse oximetry) is to shift it into the nurses hands. I don't think spot checks should be a respiratory procedure!

Rodriguez: As regards the cost versus complexity comparisons you made of monitoring equipment, did you also include the cost of the interfaceable equipment such as probes and chart and graph paper or the cost of replacements?

Ritz: It did not include operational cost. It included everything you need to get up and going with any of those devices; if you run out of disposable supplies, it is going to cost you more.

Rodriguez: Would the additional information cause you to make a different decision?

Ritz: Well, I thought about trying to look at an operational cost graph the same way, but I really found that to be even more complex than just calling up the vendors and saying give me your price range.

Krieger: Ray (Ritz), I want to comment on some of the things you said about spot checks. I actually would like to see spot checks not taken out of the hands of my respiratory care department, but rather to have my therapists be the major determiners of spot checks in an effort to decrease inappropriate oxygen use. My therapists have informed me that 50 to 75% of what they see being done on the floors, as far as oxygen therapy is concerned, isn't needed! A spot check in their hands, with certain criteria set up, could establish whether there is a need for oxygen therapy. So, that's another way of looking at it. I know exactly what you're saying when you talk about physicians or nurses asking for the spot checks. The other thing is, I think it's going to be an almost impossible task for you—or any of us—to educate all the physicians to come up with criteria for ICU admission or for oxygen use. I think that once criteria are established by selected physicians, it's up to the therapist to

implement them. I think if you rely on physicians, especially nonspecialists, to implement them, they won't understand the complexity.

Ritz: Oh, I agree! The only reason why I think the physicians must be involved is that a criterion that doesn't have sufficient medical backing is not well accepted.

Krieger: I think select physicians need to be involved.

Ritz: Oh, absolutely, I agree! I think there's stuff out there in the literature that basically says that trying to educate a large group of people—whether it be a large group of nurses, therapists, or physicians—to do the same thing, the same way (all the time) meets with miserable failure.[1,2] You end up with a wide variety of people using very different criteria.

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2. Hart SK, Dubbs W, Gil A, Myers-Judy M. The effects of therapist-evaluation of orders and interaction with physicians on the appropriateness of respiratory care. Respir Care 1989;34:185-190.

Krieger: Another very appropriate inhospital use for pulse oximetry or breathing pattern monitoring is in a sleep lab.

Ritz: I agree!

Kaemarek: I want to make a comment about doing spot checks. We just flatly refuse! We do not do spot checks, because it just became insane. The only way I believe that it would work is if physicians would write specific criteria for their oxygen therapy and then give the therapist the responsibility and authority to titrate the F_{IO_2}, to change the apparatus, and to do what needs to be done based upon the information. But, if the institution is not willing to do that—I agree with Ray (Ritz)—it's a complete joke to have people performing spot checks in response to the whim of a nurse or a physician. You just cannot do it!

Krieger: I'd like to respond to that. I agree with you! I think the criteria should be set up and the implementation should be by respiratory therapists; I think that they should run the show!

Kaemarek: But they have to be given the authority to discontinue it, to change the equipment, to do whatever is necessary to make it work within the criteria.

Krieger: Yes, you have to go through the medical executive committee. It's just like giving them the authority to change someone from a side-arm nebulizer to a spacer device for delivery of inhaled medications. I know in some hospitals, ours for instance, therapists do have this authority, and I think it's very appropriately placed there.

Branson: I have a couple of comments to make concerning metabolic carts. First, if you spend more than $30,000 you also get exercise testing and/or respiratory monitoring that can be performed in the ICU, eg, dead space-to-tidal-volume ratios and work of breathing. Though I don't think the cost is justified—I'd like to see them be much cheaper—you do get a lot more for your money than just metabolic measurements. Another comment I have is regarding the study on nutritional support published in *Journal of Parenteral and Enteral Nutrition*.[1] Only nutritional support departments were interviewed, and, of course, nutritionists are having trouble doing metabolic tests in the ICU because they don't understand anything about ventilators. I think that was the biggest reason behind those metabolic carts not being used. I hear people complaining about the complexity of doing tests with a metabolic cart, but at the same time I say "Why are you complaining? You have a system that you can use to affect patient care, that requires a competent, technically skilled person to operate—and you have just described what this profession is all about!" I think we ought to embrace metabolic testing because it is a useful monitoring technique. It's something that we can do, and it will affect patient care. Ray (Ritz), have you bought a metabolic cart recently?

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Ritz: Not because I wanted to! We bought one because we have some physicians who want to use it, and it's not clear how long they want to use it. What I would say (and I thought about including this in the talk) is the real key thing that we learned in our review of metabolic carts recently, prior to our purchase, was that if you don't make the nutritionists an active part of the team, you really lose a significant portion of a loop that makes for a quality metabolic-assessment process. I think the institutions that do a good job involve the physicians, respiratory care, and nutritional services. That has been my line to metabolic cart vendors: "Don't you dare go talk to a respiratory therapist without talking to a nutritionist also!" I think we need both of these schools involved for this technology to be useful and appropriate. I don't want you to get the wrong idea—that I don't like metabolic carts. I just think metabolic carts desperately need direction, and we need to focus on how we're going to utilize them. I think if someone doesn't have that focus, then I really would seriously counsel them not to acquire a metabolic cart. I don't think the utility of metabolic carts (that they get people up and walk them out of the hospital) is absolutely proven. People got up and walked out of the hospital before we had metabolic carts, oximeters, and all that stuff.

Barnes: I just want to reaffirm what John Marini said earlier in the conference—education, in terms of what you

do with the data that you get from these monitoring devices, is critical. When you use metabolic carts, even in a place like Massachusetts General Hospital, you're talking about one or two people (maybe three) who are going to use them primarily on research projects—correct me, Bob (Kacmarek), if I'm wrong about this. So, I think we need to focus on transcutaneous devices and pulse oximeters and how they should be used appropriately; on how to educate nurses who work in the ICUs and noninvasive monitoring units to apply the data appropriately; and on the issue of continuous monitoring being done (where appropriate) rather than spot checks, which are just a waste of time.

Ritz: I agree.

Durbin: I'm pleased to see that other people are facing exactly the same issues that I am—in fact when I get back from this conference, the nursing staff of our hospital is meeting with me on why we're not going to do spot checks. Unfortunately, our department has a couple of extra oximeters; my decision has already been made—I'm going to give them to the nursing staff! I made that decision before I came here. I'm really pleased to see that this seems to be the consensus opinion. I would hesitate to add, though, that it's really important in my mind as a physician not to limit what a physician can do—with or without the help of a respiratory care department. As a manager, I also have to accept that there are going to be clinicians who want to practice outside the realm of the average or what you might consider the consensus approach. I can't prevent people from practicing the way they want to practice. I can't control all new technology 100% of the time, or in fact no advancement will ever be made. What I can do is make it easy for those people who are practicing within the standards and make it less easy for those people who are outside. It's still the physician's right to choose the way he wants to practice; he will be the one responsible and liable. So I agree with the educational issue; I also agree with making it harder to practice outside the standard—but not impossible. I would never draw the line and say "you can't do it in this hospital." I think that's an inappropriate position for me, a manager of a department, to take.

Ritz: I don't disagree with that; if I had fleshed out my process of utilization criteria more fully, it would have done what you said, made it tough to get outside the boundaries. I don't disagree with your philosophy at all, I just think it's important that we not make it too easy to get outside the criteria.

Kacmarek: All I was saying is that a physician should be forced to spell out what the endpoint or goal of the therapy is, and then to give the practitioner the ability to achieve that goal if the practitioner is expected to monitor the application. I'm not saying to take the authority away; I just think that there are too many situations in which practitioners are asked to do things without any insight into why they need to be done. This creates enormous problems in overall application of therapy, particularly in the intensive care unit.

Giordano: I'd like to counsel against transferring spot checks to non-respiratory care personnel. I think you're taking the shortcut if you do! In terms of a cost argument, I would argue that nursing personnel are more expensive than respiratory care practitioners—at least that's what the data show. In this country, we also have a much greater shortage of nurses than we have of respiratory care practitioners. I think that if these situations are occurring in your hospitals, there might be other solutions. When I first heard about pulse oximeters, they were sold to me along with transcutaneous monitors as substitutes or partial substitutes for blood gases. Yet, I noticed that this decrease in utilizing blood gases was not listed as an advantage in any of your slides. I have to suspect that there is what they call a 'woodworking' effect—when you get a new device, the demand just comes right out of the woodwork! All of a sudden you can't live without it, and you hadn't even heard of it 24 months earlier. Perhaps the solution is to look at the basis of the demand here and get back on the track. It sounds like these devices have a legitimate role in noninvasive monitoring, but it looks like we're going about it in a way that's like a downhill roller coaster ride—it's somewhat out of control! We are perhaps just grabbing at some short-run solutions that we may regret in the future!

Ritz: Well, Sam (Giordano), to address the issue, Is it cheaper for a nurse to do pulse oximetry or a therapist? I'd say nurses might make more per hour than a therapist, but if you look at the time it takes to do a single spot check for a therapist and a nurse, the nurse's transit time is significantly less because she's right there on the floor already, whereas the therapist has to come from another area. So if you look at cost per unit of time I would wager you that it's more expensive for a therapist to do it in a non-ICU setting than it is for a nurse who is stationed on that floor to do it. I understand your concern; I'm not crazy about giving it up, but I ain't crazy about keeping it either! It's like having a handful of bees!

Kacmarek: I think that you're right—nobody likes to give up territory. But I think, in this milieu of limited resources, it's appropriate for the respiratory care department today to give up tasks that may not require the skill of a respiratory therapist in exchange for additional tasks that really do require highly skilled practitioners. Nobody's going to give us additional staff to perform all of the new types of monitoring in addition to our current work load. I mean, we just got a metabolic cart. I have to squeeze the personnel to utilize that

metabolic cart out of my existing staff; if things go the way I anticipate they will go, I will have a dedicated person in the next 6 months doing metabolic studies—period! I have to give something up, to rearrange, to become much more efficient. The assumption that we can simply add and add and add—even to keep those tasks that really do not require a respiratory therapist (registered respiratory therapist)—is absurd! I think oxygen therapy (to set up, to maintain an oxygen cannula) is probably a very reasonable thing for nursing personnel to do in preference to respiratory care personnel.

Giordano: I'd just like to counter with one thing. To deal with it the way you suggest, Bob (Kacmarek), is to acquiesce to perhaps an artificially high demand for the service! I submit that it would be more appropriate before you resort to the last resort, which is to give it up to people who are not trained to do it (such as nurses), to look at the demand first. Perhaps if this woodworking effect is addressed head-on, and that's what I think it is from what I'm given to understand here, then perhaps your practitioners would have the time. If they didn't have to do the unnecessary ones, perhaps they would have the time to do the necessary ones.

Durbin: At my institution, if the O_2 saturation comes back low, the nurse calls the therapist to evaluate the patient. So, we get involved in those patients who need to be seen by an expert. But, it really is a problem, because for every one that comes up abnormal there are 99 that are the result of a nurse who was nervous!

Mathews: Ray (Ritz), how are you determining what to charge for the oxygen the patient is using? Does everyone get charged? Does somebody do oxygen rounds to see that the oxygen is running? Who makes out the charge?

Ritz: I have a non-technical person assess oxygen utilization; we have a daily charge for oxygen.

Mathews: They go around and make sure that the patient is using it? Why can't they do a pulse oximetry measurement then? That would solve the problem of using a therapist, since it's a non-technical person, and oximetry stays within the department.

Ritz: Well, if we had to check everybody who is on oxygen with pulse oximetry, we would be doing even more spot checks than we are currently!

Mathews: Well, you wouldn't have to do that—you could say that you do spot checks during a certain period of the day, and that's it. I mean, oximetry for this purpose is something that can be scheduled.

Ritz: Well, the things that we've done to control pulse oximetry have been: We've restricted continuous oximetry utilization to intermediate- and intensive-care units only (we don't put a continuous monitor where nobody's listening to it); we require that all spot checks done by respiratory care be in response to an individual written order (no q4h, or qAM, but every time a physician wants oximetry he must write an order); any saturation below 90% precipitates a phone call to the physician. The last thing we did was to initiate 'oximetry rounds.' We put little boxes out on all the floors into which nurses drop carbon copies of the orders, and we go around at 7:00 in the morning to pick them up. Then we do the oximetry checks—once a shift.

Mathews: If you've got unnecessary ones, though, the thing to do is not just call the physician when it's below 90%; call him on every one he orders! If you call him 50 times a day it could slow him down—make him think about and reassess his ordering pattern!

Ritz: Well, on nightshift we call every time—we figure if a physician wants oximetry done at night then he must want the information! More than once we've awakened a physician who responded by saying, "but I never ordered that!" What we realize then is that we're treating nurse anxiety. What we've done is that we have just tried to make the criteria a little bit tighter by putting a few more strings on the process, but our bias is to get rid of this.

Noninvasive Monitoring in Respiratory Care: Conference Summary

Robert M Kacmarek PhD RRT

As Dean Hess and I developed this conference, our goals were: (1) to completely cover the topic of noninvasive respiratory monitoring; and (2) to select participants with diverse opinions to stimulate discussion. I believe we have achieved these goals if by no other measures than the more than 50 pages of notes that I accumulated over the two days of the conference and the lively discussions that followed each presentation. I congratulate all of the participants on a job well done.

I would like to approach this summary by going back to each of the presentations to briefly emphasize the key points. I will complete the summary with my perspective on the whole subject of noninvasive cardiopulmonary monitoring and will highlight the message that I hope this conference sends to the respiratory care community.

Dean Hess began the conference by presenting an overview of monitoring in respiratory care. He defined a number of terms and provided a historical overview. One of the things that he did best was to put into perspective what is meant by monitoring. He defined monitoring as the continuous assessment of a variable. Even if it is periodic in determination, monitoring must continue over time and provide real-time information to be considered monitoring. He also outlined some of the reasons why there has been such a proliferation of noninvasive monitoring: (1) the desire to avoid invasion of the patient because of the potential for infection and the effect invasive techniques may have on results; (2) the desire to provide continuous real-time data; and (3) the role of the manufacturer—his perception (and mine) is that industry has played a significant role in the proliferation of noninvasive monitoring techniques. In an attempt to define how much monitoring is really needed, he raised more questions than he answered—Is more really better? Unfortunately, in many of our institutions *more* is the rule rather than the exception. Do practitioners understand all of the data presently available? In many cases I would answer this question with a resounding No! As a result, appropriate utilization and objective evaluation of the need for information is lacking. Dean also emphasized that frequently because of the number of monitors employed, the monitors themselves become distractions. They create a data overload and increase the probability of false-positive alarms. The result—practitioners tend to ignore alarms. I know that, in my own institution, if I sit at a nurse's station in the ICU and wait for an alarm to sound and use a stopwatch to determine the time it takes somebody to respond, frequently it may be 30 or 40 seconds because they have previously responded to the same alarm, with every single alarm proving to be a false-positive.

Dean also emphasized that no data relate the implementation of monitors to outcome and limited data deal with the financial impact of specific monitors. We all assume that the introduction of an additional monitor improves efficiency and reduces cost. I'm not sure that is the case. As a matter of

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fact, I would argue that the introduction of a new monitor often results in increased cost instead of a cost savings because it is rare that we eliminate an old monitor as we add a new one. We simply increase the number of variables monitored. Pulse oximetry, for example, has increased the number of blood gases analyzed in many institutions instead of reducing the frequency.

Next, Tom East provided us with an overview of the basic engineering principles associated with noninvasive monitoring. He discussed those techniques utilized to evaluate flow, volume, pressure, gas composition, gas exchange, and hemodynamics but then focused on relating computer applications to monitoring technology. He indicated that a gap commonly exists between the engineering community and the end-user community in the overall development of any monitor, and, as a result, presentation of data is frequently not friendly to practitioners. Tom highlighted the need for some standard format, some "communication bus" adopted by all companies so that monitors could talk to one another, allowing us to make more sense of data. This would allow data to be processed by a central system that filters, organizes, and presents us with a refined version of the monitored data in a format more useful and much easier to interpret.

Tom went on to challenge us by saying that one of the problems that the engineering community has is that we (the respiratory care community) have no standards associated with monitoring. Nobody has defined what is important, what needs to be monitored, what does not need to be monitored, what variables are critical, and what variables are noncritical.

Following Tom's presentation, Rob Chatburn took us through a detailed analysis of techniques used to assess monitors. His key point was to question the ability of monitors to perform as stated. He mentioned at least four types of errors common in the evaluation of instrumentation and emphasized that the extent of an instrument error can be easily hidden in the presentation of data by manufacturers. He cautioned us to carefully evaluate the manufacturer's performance data.

He then discussed standards and methods that are used for comparison, emphasizing the lack of a 'gold standard' for accuracy of monitoring. As a result, standards in this area are developed by professional consensus or by arbitrary statistical inferences. Therefore, the likelihood of error in techniques based on these standards is much greater than in those approaches for which a true gold standard for comparison exists. He went on to state that the fundamental questions in evaluating equipment are: (1) Is the accuracy of the equipment acceptable for the intended use? (2) How close will a single measured value be to the actual value that it is designed to measure? (3) What should be considered when one compares two techniques to measure the same variable? Rob went on to discuss the determination of agreement between two measurement techniques. He stressed that an acceptable method is to plot the mean differences between the two measurements against the average of the two measurements. Those values falling within two standard deviations of the mean differences represent limits of agreement. He also stressed evaluation of the specific level of variance within those two standard deviations and to evaluate for ourselves whether that variance is acceptable clinically before we assume that the instrument is reliable and precise.

Following Rob's presentation, Neil MacIntyre presented information regarding 'low-tech' monitoring. I found Neil's presentation particularly interesting because it is the kind of monitoring that I believe most respiratory care practitioners, nurses, and physicians have neglected and, as Neil emphasized, the monitoring that all of us need to perform daily. He reviewed the standard physical assessment skills of inspection, palpation, percussion, and auscultation, and tried to define how adequate they are in their determination of basic alterations in physiology. In this discussion, he emphasized that the accuracy, reliability, calibration, ease of operation, and function of low-tech monitoring as an early warning sign is totally dependent upon the practitioner's clinical capabilities (that is, how well the practitioner maintains and develops those clinical capabilities). The points Neil made are excellent because the biggest single issue that I see as we continue to integrate 'high-tech' methods of monitoring is a lack of the practitioner's perceived need to maintain these low-tech skills. Neil listed five aspects of pulmonary function to be assessed: gas exchange, ventilatory drive, airway function, pulmonary vascular function, and ventilatory muscle function—and contrasted low-tech and high-tech

approaches to their evaluation. For the assessment of ventilatory drive, overall airway function, and ventilatory muscle function, low-tech monitoring exceeds the capabilities of high-tech monitoring. Neil's discussion emphasized one of the key points of this conference—in spite of the fact that we have monitors that can check a myriad of variables, it is still essential for practitioners to maintain, develop, and use their physical assessment skills.

Dean Hess followed with a discussion of capnography. He began by defining the difference between capnometry and capnography and discussed in detail the overall operation of different units. The key point of this presentation is the fact that the end-tidal CO_2 is not necessarily a direct reflection of arterial P_{CO_2} and, in fact, these two variables can change in opposite directions. Dean stressed that end-tidal CO_2 is more specifically an indicator of cardiopulmonary homeostasis than it is an indicator of the overall level of ventilation. For example, the patient breathing rapidly and shallowly, who is also in cardiovascular failure may demonstrate a decreasing end-tidal CO_2, with an increasing arterial P_{CO_2}. He went on to indicate that end-tidal CO_2 can vary from as low as the inspired CO_2 to as high as the mixed-venous P_{CO_2}. As a result, in patients in whom a number of organ systems are malfunctioning, the reliability of the end-tidal CO_2 is questionable. Therefore, I agree with Dean's assessment that end-tidal CO_2 monitoring has a limited role in the monitoring of patients outside the operating room and, in fact, may provide misleading information. At this stage in the development of this technology, it is useful for confirming tracheal intubation and functions well as an expensive, sophisticated ventilator-disconnect alarm.

Richard Martin followed with a discussion of transcutaneous monitoring, an approach to monitoring that has taken a back seat to oximetry in many neonatal intensive care units. Richard emphasized that transcutaneous monitoring is more precise than pulse oximetry in tracking P_{aO_2} changes in premature infants in whom P_{O_2}s are normally maintained in the 60-80 torr range.

He went on to state that the primary advantage of monitoring transcutaneous partial pressure of oxygen (P_{tcO_2}) rather than pulse oximeter saturation (S_{pO_2}) is in preventing hyperoxia and its associated problems. He also finds P_{tcO_2} helpful in quantifying or at least identifying the presence of a patent ductus arteriosus. The limitations associated with transcutaneous monitoring and probably those that have minimized its use more than any other factors are (1) technical complexity; (2) electrode temperature requirements (43-44 °C); (3) skin-burn problems; (4) need for changing electrode location; (5) alteration of transcutaneous readings by poor perfusion; and (6) use limited to infants less than 8-10 weeks of age because as infants grow the accuracy decreases. Transcutaneous O_2 monitoring is less reliable in the adult, although there is fairly good agreement between P_{aCO_2} and P_{tcCO_2} in the adult population. However, neither technique has gained widespread acceptance in adults.

Jim Welch followed with a discussion of pulse oximetry, the one approach to noninvasive monitoring that has inundated all of our institutions with orders for implementation to a greater extent than the other techniques addressed in this conference combined. Jim began with a detailed discussion of the historical development and technical operation of oximeters, then presented those variables that affect performance, accuracy, response time, and amplitude of the signal. In addition, Jim stressed that the initial calibration performed by the manufacturer is empirical and that *no routine calibration* is performed by the practitioner.

Jim went on to indicate that the ear probe generally produces more accurate results than does the finger or toe sensor. He also stated that the accuracy of these instruments for clinical trending is very good at saturations above 85%, is fair at saturations above 70%, but is poor at saturations below 70%. However, the specificity of the measurement is very poor at saturations between 95-100%. This, coupled with an overall variance of measurement between $\pm$ 2 to 4%, enhances the concern regarding hyperoxia in the neonatal population with saturations $\geqslant$ 95%. He also stated that the use of oximetry has been mandated in the operating room (O.R.). In our institution and in most that I have visited, oximeters are attached to every anesthesia machine. Any time general anesthesia is administered, an oximeter is attached to the patient. Jim believes that this is primarily the result of critical-incident tracking and the hypoxic episodes that have occurred as a result of critical incidents in the O.R. Even though the cost of adding oximeters to every anesthesia machine is high, it is considered much less expensive than just one lawsuit

resulting from a hypoxic episode. I would thus list liability as the primary reason for pulse oximetry use in the O.R. Jim cited 1.7 problems or critical incidents per 10,000 cases. Unfortunately, no such data are available in the intensive care unit (ICU).

Because oximetry provides valuable trending information in certain clinical settings, it tends to be more misused and overused than any other technique presented at the conference. The two major areas of abuse, from my experience, are in the neonatal ICU and in the adult ICU for the patient being weaned from mechanical ventilation. In the neonatal ICU, saturations maintained in the 95-100% range increase the probability of hyperoxia. In patients being weaned from mechanical ventilation, oximeters are commonly used as the only indicator of failure. Attention to clinical signs of inadequate muscle function and cardiopulmonary stress are frequently lacking and, of course, the erroneous assumption that because the saturation is above 90%, gas exchange must be acceptable. As a result, I would caution against the use of oximetry instead of transcutaneous oxygen monitoring in neonates and would advise against the use of oximeters during weaning unless we can correct the profound educational deficit regarding the limitations of this noninvasive monitor.

Richard Branson next discussed indirect calorimetry. Rich began his presentation by making an argument for the need to perform indirect calorimetry. He stated that it was a reasonable method of guiding nutritional support, monitoring the appropriateness and the efficacy of nutritional support, avoiding complications associated with over- or underfeeding, and evaluating substrate utilization. He also stated that even though in his mind these instruments are very useful, they present technical problems: (1) They are difficult to calibrate. (2) The procedure requires that the inspired oxygen concentration be precisely maintained. (3) They are relatively complex and time consuming to use. (4) They are costly to purchase. Rich went on to detail the problems associated with the different types of systems (open versus closed-circuit) and the difficulty in interfacing mechanical ventilators with these instruments in the ICU.

Rich also discussed concerns that he had regarding the adequacy of utilizing the normal equations for estimating nutritional support. In his opinion, the Harris-Benedict equation, even with its modifications, results in over- or underestimation of the nutritional needs of many patients. He went on to state that he felt that any patient maintained on mechanical ventilation for longer than 7 days can benefit from nutritional assessment.

It is difficult to argue with Rich regarding the need for more precise estimates of nutritional needs. However, because of the profound difficulties in arriving at accurate data in ventilated patients and the fact that indirect calorimetry, in our experience, rarely impacts overall outcome, I do not believe the average patient's care is adversely affected by a lack of application of this technology. If nutritional support is delivered conservatively, we rarely find that indirect calorimetry has any impact other than to add to our knowledge base.

David Pierson then gave us his impression of lung volume determinations outside of the pulmonary function laboratory. Dave focused primarily on evaluating FRC. He emphasized that FRC evaluation can be very useful: (1) in patients with adult respiratory distress syndrome; (2) in those patients where there is a concern about overdistension and consequent barotrauma; (3) in the COPD and status asthmaticus patient, in whom air trapping is suspected; (4) as a method of detecting and monitoring complications of ventilator care; and (5) as a means of assessing the effect of position changes on overall lung volume. After saying that, he went on to discuss the possible techniques: open-circuit, closed-circuit, radiographic, and respiratory inductive plethysmographic means of measuring changes in FRC. He indicated that although all of these methods are feasible, they are extremely difficult technically and are probably not reasonable to perform on a frequent basis outside of the laboratory.

Dave then went on to make an argument against the measurement of FRC in the ICU, saying that because most lung disease is nonhomogenous, what does the FRC level actually indicate or how can we use it even if we have it available? He concluded by saying that, Yes, we would like to measure FRC in spite of the problems with interpreting the data, but at this time we probably do not have any reasonable systems capable of measuring FRC. Immediately afterward, Tom East informed us that in the very near future a new system that is being developed by Siemens will be available. This system, according to Tom, is relatively simple, straightforward, easy to use, and may change our whole attitude

toward monitoring lung volume in the ICU. Although Tom's comments are scientifically exciting, I do not believe, as Dave indicated, that there is sufficient information at this time to intelligently use FRC data in day-to-day management decisions. We do hope that this new technique will begin to provide data to guide clinical management.

John Marini next discussed lung mechanics. He emphasized the need to carefully monitor airway pressure; however, in spite of the availability of airway pressure data, John believes airway pressure is one of the most underutilized variables that we have at our disposal. He went on to state that airway pressure is key in determining how effective the ventilator setting is in meeting a patient's ventilatory demands and in providing a means of assessing patient effort and ventilatory drive.

John also discussed the merits of measuring maximum inspiratory pressure and indicated that if the measurement is performed properly it provides valuable information regarding changes in ventilatory muscle capabilities, over time. Following this, John discussed auto-PEEP—its measurement, its significance, and the circumstances in which auto-PEEP occurs. John also pointed out that the use of external PEEP to modify the effect of auto-PEEP should always be employed with caution. He believes that it is reasonable to use PEEP for this purpose only in those patients in whom auto-PEEP is a result of airflow limitation. He then commented on the need to monitor compliance, resistance, and the work of breathing, indicating that these measurements can be determined at the bedside, as long as we can evaluate a few passive mechanical volume-limited inspirations.

Bruce Krieger discussed respiratory inductive plethysmography (RIP). He emphasized that most pulmonary catastrophes can be identified prior to their occurence by alterations in ventilatory pattern and ventilatory rate. Bruce described the use of RIP systems to monitor thoracic and abdominal excursion and to identify dyssynchrony. He also emphasized the complexity of the available systems and the educational needs of the end user.

Bruce went on to define those circumstances in which he felt that RIP was useful (ie, pulmonary function laboratories, sleep centers, and as a method for noninvasively monitoring patients who are mechanically ventilated or spontaneously breathing patients about whom there is a concern for ventilatory failure). He indicated that it would be reasonable for all of us to consider stepdown units or stepup units for the monitoring of ventilator-dependent patients who are not critically ill. He ended with a detailed discussion of fiscal concerns and potential cost savings in developing these types of units.

All would agree that RIP is a useful research tool; however, it appears that few believe it to be a necessary clinical monitor. This assessment is based partly on the complexity of available systems, cost, and the fact that properly performed low-tech monitoring provides exactly the same overall information as RIP, although in a qualitative manner. I highly endorse Bruce's concept of a stepdown ventilator care unit; in fact, we have a number of these units associated with different medical services, but I strongly disagree with the need for RIP to ensure appropriate management.

Next, Charles Durbin discussed approaches to noninvasive hemodynamic monitoring, focusing on two areas: evaluation of blood pressure and evaluation of cardiac output. He began by discussing those variables that affect the likelihood of identifying an event considered necessary to monitor. Specifically, he listed four factors: (1) the frequency of the event; (2) the duration of the event; (3) sensitivity of our monitoring systems; and (4) the frequency at which the sample is obtained. He went on to discuss the different approaches utilized for noninvasively evaluating blood pressure and indicated that these devices may not be comparable to direct invasive measurement of pressure. Direct invasive approaches measure pressure, whereas indirect approaches measure alterations in flow. He went on to state that most noninvasive systems are acceptable for trending changes, although they frequently underestimate hypertension and may overestimate hypotension.

Following this, Charlie discussed cardiac output monitoring, indicating that available methods are relatively inaccurate—although, again, they may be useful in trending of data in select population groups. He categorized patients according to their associated cardiovascular monitoring needs: (1) stable outpatients—simple blood pressure measurements; (2) patients suffering from a myocardial infarction (MI), but relatively stable—noninvasive approaches to blood pressure monitoring; (3) MI patients with questionable stability—invasive monitoring of both blood pressure and cardiac output; and (4) MI patients

who are grossly unstable—invasive monitoring of blood pressure and cardiac output and noninvasive monitoring of cardiac output.

I then discussed the use of noninvasive monitoring outside of the hospital. Little or no data are available to guide the practitioner in the application of any of the monitors discussed—outside the hospital. In addition, no routine reimbursement exists for monitoring outside of the hospital setting. No consensus on indications for monitoring has been achieved by practitioners in centers involved in home care, and no clear indications for the use of any type of noninvasive monitor have been published in the home care literature.

I then went on to discuss the use of oximetry on a periodic 'spot-check' basis to trend patients receiving oxygen therapy once the need has been established by blood gas analysis. I also emphasized that no indication exists for the continuous oximetry monitoring of adults in the home. Those rare circumstances in which oximetry has been used continuously in the home have been in infants. The use of continuous end-tidal CO_2 monitoring during mechanical ventilation also has a limited role in the home care setting. Periodic assessment with oximetry, low-tech monitoring, and ventilatory mechanics monitoring are encouraged whenever practitioners visit patients on continuous mechanical ventilation. However, in my experience, there is rarely, if ever, a need for continuous monitoring of anything other than the ventilator system, in the home care setting.

The overriding concern that I hope to express is the lack of information about noninvasive monitoring in the home. No guidelines and no standards exist. A consensus conference to clearly define who, when, how, and why we should monitor patients outside of the hospital setting is needed.

Ray Ritz made the last presentation, providing a well-organized review of the indications for most of the techniques discussed. He told us why he believes that noninvasive monitoring is overutilized. First, expectations that the data will significantly alter or assist our management of patients; second, increasing litigation; third, improved and easier-to-operate pieces of equipment in comparison to that previously available; and finally, data for the sake of data. Most of us like to look at numbers, many of us are involved in research where the more variables we have to assess, to interpret, to interrelate, the better we feel. Whether those variables really have an impact on the overall care of the patient frequently appears to be irrelevant.

Ray discussed those factors that dictate the number of devices needed by an institution. In general, he indicated numbers are based on bed size and overall mix of patients—how many ICUs, how many stepdown units, how many recovery-room beds, and the type of specialized units. Staffing was another concern—whether the introduction of the new technology resulted in improved efficiencies or a need for additional staff; and finally, the availability of financial resources.

Ray's presentation ended with a summary of strategies for resource conservation, with a focus on the need to ensure appropriate utilization of noninvasive techniques. The first point on his list was education—education of physicians, nurses, and therapists. He went on to discuss participation in purchasing decisions. Too often a technology is introduced and nobody has any idea of the problems associated with its use or what the data mean. He discussed the need for medical audits, for utilization reviews, for observation and review of overall practice (once a technology has been introduced) and the establishment of criteria, guidelines, and indications for initiation and discontinuance of specific monitors. Finally, Ray emphasized that the influx of new technology should be minimized to ensure careful evaluation and determination of clinical efficacy before purchase of a new piece of equipment.

Now let me take a few moments to further editorialize and provide my concluding remarks. Many of my comments may be perceived as being antagonistic toward new monitoring techniques. I am not opposed to new monitoring approaches, but I am most concerned about the inappropriate use of new technology. Noninvasive monitoring technology is a necessary addition to patient assessment and will continue to grow and develop. I believe it improves overall patient care when appropriately utilized. However, my concerns primarily involve inappropriate utilization and utilization simply because you have the monitor—collecting data simply for the sake of data. In my opinion, two forces are driving inappropriate and acritical utilization: marketing and litigation. In many cases, we add monitors simply because we are afraid of missing an event that *may* precipitate a lawsuit—and I'm not sure that is an

appropriate reason for adding technology to our overall medical care armamentarium. Advances in technology, of course, are going to allow us to introduce additional monitors and expand our capabilities. I believe everyone who is a champion for any of these devices has enhancing the patient's well-being in mind; however, I think we misinterpret the capability of others to utilize a given technology or the global need for a technology.

What are my concerns? The same concerns that have been addressed at this conference—primarily, *education*. If there is one point that I would like to bring home in this editorial, it is that we need to ensure that the respiratory care community is appropriately educated in the use of present monitoring systems and prior to the introduction of new technology.

Second, I think that we do need to work with the engineering community to come up with better methods of presenting the data. Systems have to be more user friendly, if for no other reason than because there are more of them to use. At the average ICU bed, there are 10 or 12 devices that the bedside practitioner must be fully capable of operating and fully capable of responding to. Many complex systems are not being utilized appropriately or to their maximum capacity.

Third, alarms are a critical problem. Manufacturers tell me because of liability they cannot give us the flexibility to inactivate an alarm on a machine. If this is the case, then we need smarter alarms. We need alarms that are not activated by a momentary drop in saturation but must exceed a threshold for a period of time or have some intelligent built-in algorithm that ensures that the frequency of false-positive alarms is drastically reduced. As a result of the alarm status in most of our ICUs, practitioners are programmed to ignore alarms! If you watch practitioners respond to alarms, you will see that they don't evaluate the equipment, they don't evaluate the patient. They simply press the reset button, turn around and walk away from the bed! To me that's a critically unsafe situation, and I believe the proliferation of false-positive alarms intensifies this problem.

Fourth, criteria need to be established for the use of each monitoring system. The attitude that because the monitor is available it should be used must change. The accumulation of data simply for the sake of data must end. We have to more intelligently utilize the systems that we have available.

Finally, my overriding concern is that, as a result of increasing numbers of monitors entering the intensive care area or the hospital in general, I see practitioners spending less and less time looking at patients, touching patients, and listening to patients. I find this distressing because, as Neil emphasized, we can accumulate so much data with our senses that are not available from monitoring devices. We are doing patients a grave disservice if we allow the continued introduction of monitoring devices to focus more and more of our attention on equipment and less and less on patients.

Books, Films, Tapes, & Software

Hemodynamic Monitoring, edited by Jacqueline Loach RN MA and Norman N Thomson Jr MD. Paperback, 178 pages, illustrated. Philadelphia: JB Lippincott Co, 1987. $15.50.

Written primarily for critical care nurses new to the procedures involved in hemodynamic monitoring, this book could also be used as a quick reference by more experienced practitioners. Facets of hemodynamic monitoring discussed include nursing activities surrounding catheter insertion, maintenance, and removal, and interpretation of normal and abnormal hemodynamic waveforms, pharmacologic influences on hemodynamics, nursing management, and standards of care for monitored patients, and techniques for orientation of new personnel to the practice of hemodynamic monitoring. Chapter authors include the nursing care coordinator of a cardiac SICU, cardiovascular clinical nurse specialists, an ICU staff nurse, and the chairman of a department of cardiothoracic and vascular surgery.

Unfortunately, the first chapter of the book, an overview of the subject, could be confusing to persons unfamiliar with hemodynamic monitoring because of some inaccurate terminology such as "left atrial wedge pressure" (Page 5) and "capillary pressure catheter" (Page 6) and unclear tabular information (sodium nitroprusside and nitroglycerine neither of which is an inotrope included in a table titled "Inotropic Drugs and Drug Combinations" on Page 10).

Subsequent chapters are more clearly written and easy to read, but due to rapid advances in technology much of the information is out of date. For example, nine pages are taken up with the techniques of assembly of transducers and associated tubing when nearly all hospitals have converted to pre-assembled disposable systems. In addition, some practices recommended in the book such as placing the patient in a supine position for monitor readings and having the patient in the same position for all readings have been shown by recent nursing research[1,2] to be unnecessary and unduly stressful for the patient.

Inconsistencies in the text may also prove confusing to the inexperienced practitioner. The amount of heparin to be added to I.V. flush solution, for example, is variously stated as 500 units (Page 27) and 2000 units (Page 30).

The chapters on nursing management of and standards of care for the monitored patient and the chapter on factors that influence hemodynamic values are well done and contain much valuable information.

Over all, this book provides useful information on the basic procedures involved in hemodynamic monitoring, but would require supplemental material on recent technology and procedural research to adequately cover the subject.

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Medical Management of the Cardiac Surgical Patient, edited by Richard J Gray MD and Jack M Matloff MD (with 27 contributors). Hardcover, illustrated. Baltimore: Williams & Wilkins, 1990. $52.95.

Open-heart surgery, that is, cardiac surgery performed with extracorporeal circulation, has become commonplace in community hospitals, and in specialized centers 30 or 40 of these procedures may be performed daily. The respiratory care practitioner is usually involved with the postoperative care of these patients, most of whom are ventilator-dependent for at least a few hours. If serious complications develop the respiratory therapist may be a central figure in the resuscitative effort. A comprehensive understanding of cardiopulmonary bypass and its implications for postoperative respiratory function is essential to intelligent appraisal and effective care.

This 378-page book is a substantial compendium, covering all aspects of medical management of patients undergoing open-heart surgery. Of the 27 contributors, 19 are from the Cedars-Sinai Medical Center in Los Angeles, California. A foreword by HJC Swan introduces the theme of the work, defining the appropriate division of labor between cardiologists and members of the surgical team. The book is divided into 6 sections. The first of these is an overview of the development of cardiac surgery that outlines the present indications for coronary bypass and valve replacement. The remaining sections parallel the course of the patient through the preoperative, intraoperative, postoperative, and late follow-up periods.

Section 2 covers the preoperative workup, including the assessment of risk. A special chapter is devoted to the psychological preparation of the patient and family. Section 3 deals with the intraoperative management of the

patient and is written mainly by anesthesiologists. The first chapter of this section is of especial interest to the pump technologist; it concerns the operational aspects of cardiopulmonary bypass.

Section 4 encompasses the early postoperative phase. It is not only the largest portion of the book, but also the part most interesting and potentially useful to the respiratory practitioner. Although only one short chapter is devoted specifically to pulmonary care, the entire section contains much information of immediate importance in respiratory management. Low cardiac output states, hemodynamic changes, bleeding diatheses, and arrhythmias are situations in which the accurate monitoring of gas exchange is essential to the establishment of stable heart function. These subjects are covered fully, with many practical recommendations for optimizing ventilation and oxygenation. The authors of these chapters are clinically experienced, and highlight their discussions with opinions that reflect firsthand knowledge of these complications.

Sections 5 and 6 are concerned with problems encountered after the patient leaves the hospital. These chapters are informative but will be useful only to respiratory practitioners engaged in home care.

Most of the sections are well written and appropriately referenced. Illustrations, tables and charts, while not numerous, are well chosen. Editorial proofreading should have been done more carefully; in some chapters, typographical errors average more than one per page. Overall, the book should be amply rewarding to respiratory practitioners employed in tertiary care hospitals, and is especially recommended to therapists working in cardiothoracic intensive care units.

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Making Sense of Data: A Self-Instruction Manual on the Interpretation of Epidemiological Data, by JH Abramson. New York: Oxford University Press, 1988. $39.95.

According to the author (who is Professor and Chairman, Department of Social Medicine, the Hebrew University-Hadassah School of Public Health and Community Medicine, Jerusalem, Israel), the purpose of this book is to provide readers with basic epidemiologic concepts and skills that will help them to appraise published reports as well as their own findings. The book includes considerations and examples in clinical medicine, public health, community medicine, and research; however, it is heavily weighted towards public health and community medicine.

The book is divided into five sections: Basic Concepts and Procedures, Rates and Other Measures, How Good Are the Measures?, Making Sense of Associations, and Causes and Effects, with many units within each section. References (136 in all) are grouped together at the end of the book.

The format of the book is that of a workbook, with text and exercises intermingled throughout. Typically, questions or exercises are followed by discussion of the solutions. (I personally had difficulty becoming comfortable with this style.) A self-test is found at the end of each section.

This is not a statistics textbook. It does not cover aspects of statistics such as data-processing techniques or advanced epidemiologic methods. The intent of this book is to help the reader interpret and use data related to health care; it does not teach statistical analysis. This book is most useful for readers who have a basic understanding of research and statistics. When one goes through the exercises in this book, it may also be helpful to have a comprehensive statistics textbook handy. Because the book does not teach statistical analysis, the reader may need a statistics textbook to appreciate in more depth the information covered and the correct mathematical calculations. I am reasonably comfortable with basic statistical procedures; however, in areas where I am less comfortable (such as with

INFORMATION

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the Kappa statistic, as I discuss below), I needed to consult other reference books to understand what the author was describing. In virtually every topic covered in the book, I found myself wanting more information to satisfy my interest.

I think this book is useful but simplistic; I find that basic facts necessary to understand some of the exercises are not provided. No subject is covered in the depth that it would be covered in a statistics textbook. Many subjects are introduced but are not covered comprehensively. For example, the book discusses statistical significance, but does not discuss statistical significance tests such as the *t* test, analysis of variance, chi-square, and Wilcoxon. To the statistically naive, a more comprehensive book will be necessary for the complete 'picture.' Also, sometimes solutions to exercises are provided without adequate explanation (almost a 'trust me' approach). For example, the author discusses Kappa, a statistical test used as a measure of agreement. However, not enough basic information is provided to allow the reader to complete the exercises and arrive at the correct answer. I found it frustrating when I had to consult another statistics book to learn enough about Kappa to work my way through the book. Overall, I find the book readily understandable for material with which I am familiar; on the other hand, it is difficult to understand the material presented with which I am less familiar.

I think this book will be most useful for those with more interest in public and community health than in clinical medicine, and for those with some knowledge of statistics who would like a refresher or overview of basic epidemiologic concepts.

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CRCE through the Journal

The second annual edition of *CRCE through the Journal*—a way for members to gain continuing education credit (CRCE) by careful reading, thoughtful study, and test completion—appears in the following pages.

The 50-item test is based on papers published in RESPIRATORY CARE from July 1989 through June 1990. The issue and page numbers of the paper on which a question is based are shown in brackets following the question. You are free to consult the cited paper as you complete the answer sheet; however, best retention of information will probably occur if you read the paper in its entirety and then answer the questions.

Remember *CRCE through the Journal* is an honor system—and the benefit you gain will be in direct proportion to the effort you invest.

Instructions

1. Enter your name and AARC membership number on the answer sheet provided with this issue of the Journal.
2. Only original Answer Sheets will be graded. No facsimiles or photocopies will be accepted. 35 or more correct answers provide 6 hours of CRCE credit.
3. Use ONLY a #2 soft-lead pencil.
4. Mark only ONE answer for each question.
5. MAKE DARK MARKS.
6. If you must change your answer, ERASE COMPLETELY.
7. in the September 1990 issue of RESPIRATORY CARE.) No scores will be available from the AARC until 1990 CRCE Transcripts are released in early 1991.
8. Fold Answer Sheet on the lines indicated.
9. Place Answer Sheet in a stamped #10 envelope and mail to:

   CRCE through the Journal
   11030 Ables Lane
   Dallas TX 75229

10. Deadline is August 15, 1990 (POSTMARK). Responses postmarked after August 15 will NOT be processed.
11. Failure to follow instructions may cause technical problems that will disqualify you.

Please note that the acceptance of ***CRCE through the Journal*** credits for the fulfillment of continuing education requirements in states in which respiratory care practitioners are licensed depends solely on the specifications of that state's licensure law.

Choose the single, most correct answer.

The issue and page number of the paper to which the question applies are given in brackets [ ] following the question.

1. The following statements are true:
   a. As a group, volume-measuring devices are more likely to meet ATS recommendations for accuracy than are flow-measuring devices.
   b. As temperature increases, gas volume increases and gas viscosity decreases.
   c. Pressure-differential pneumotachometers determine flow by measuring the pressure change across a resistance device.
   d. all of the above
   e. a and c only

[July 1989:571-585]

2. The following statements are true:
   a. With closed-circuit helium dilution there is a tendency to underestimate FRC in patients with poorly ventilated lung regions.
   b. With body plethysmography and open-circuit nitrogen washout there is a tendency to overestimate TGV in severely obstructed patients.
   c. The only direct method of TLC determination is radiographic.
   d. all of the above
   e. a and c only

[July 1989:586-596]

3. The following statements are true:
   a. Measurement of thoracic gas volume (TGV) in infants requires placing only the head of the infant inside the plethysmograph.
   b. "Squeezing the wheeze" is a slang term for pediatric pulmonary function testing in general.
   c. In infants, CVC refers to the mean exhaled volume measured during a feet 'flicking' episode.
   d. Lack of patient cooperation is not a limiting factor in pediatric pulmonary function testing.
   e. a and b

[July 1989:597-610]

4. The following statements are true:
   a. Pulmonary function tests appear to be incapable of detecting asymptomatic airflow obstruction.
   b. Longitudinal experience suggests that small airways measurements are inadequate predictors of future clinically important airflow obstruction.
   c. One of the benefits of screening smokers for airflow obstruction is that abstinence is greatly enhanced.
   d. a and c
   e. a and b

[July 1989:611-625]

5. The following statements are true:
   a. The $\pm$ 20% method of selecting lower limits of normal is acceptable as a rough guideline for FEF_{25-75} and instantaneous flows.
   b. Vital capacity is known to be lower in Blacks and Indians than in Caucasians.
   c. There is no one recommended set of pulmonary function prediction equations applicable to all laboratories and patient populations.
   d. all of the above
   e. a and c only

[July 1989:626-637]

6. Pulmonary function tests differ from clinical chemistry laboratory tests in that:
   a. The measured values are highly dependent upon adequate understanding and cooperation of the patient.
   b. The measured values are unaffected by patient effort.
   c. A single range of predicted values is seldom appropriate for all patients.
   d. a and c
   e. c and b

[July 1989:638-650]

7. The following tests have been standardized by the American Thoracic Society (ATS):
   a. body plethysmography
   b. $D_{LCO\text{-}sb}$
   c. exercise testing
   d. pulse oximetry
   e. a and b

[July 1989:651-660]

8. The following statements are true:
   a. Pulmonary capillary blood volume is the major determinant of the diffusing capacity of the lungs (D_{LCO}).
   b. Diaphragmatic activity is diminished during sleep much more than somatic muscle activity.
   c. Thickness of the alveolar wall is the major determinant of the diffusing capacity of the lungs (D_{LCO}).
   d. a and b
   e. b and c

[July 1989:661-669]

9. In the Hess et al study of medications delivery performance
   a. the performance of 6 models of small-volume nebulizers was compared.
   b. the amount of medication delivered from the nebulizer was directly measured.
   c. the nebulization times for most of the nebulizers were acceptable when the diluent volume was 3 to 4 mL and the flowrate was 8 L/min.
   d. particle size was evaluated and found to be smaller at a flowrate of 8 L/min than at 4-6 L/min.
   e. b, c, and d

[August 1989:717-723]

10. The results of the study by Lawler et al suggest that
    a. the Ohmeda 3700 pulse oximeter accurately estimates HR at workrates that produce HR $<$ 160 beats/min.
    b. a pulse oximeter's inability to precisely estimate HR during heavy exercise may be the result of motion artifact.
    c. the plethysmographic waveform display assists clinicians in judging the quality of the pulse oximetry reading.
    d. all of the above
    e. b and c only

[August 1989:724-727]

11. Pulmonary thromboemboli
    a. can result from local venous stasis in an extremity.
    b. are diagnosed and treated in approximately 600,000 people in the U.S. annually.
    c. result in the death of approximately 400,000 people in the U.S. annually
    d. frequently occur in individuals who engage in physically strenuous activities on a regular basis.
    e. a, b, and c

[August 1989:728-730]

12. A congenital bronchobiliary fistula (CBBF)
    a. is a common anomaly seen in premature infants.
    b. results from the joining of an accessory bronchus and an aberrant pancreatic duct.
    c. has presenting signs that include bilious vomiting and emphysema.
    d. a and b
    e. b and c

[August 1989:731-733]

13. Choose the single most correct answer.
    a. The HIV organism has been cultured from both saliva and alveolar fluid samples.
    b. There is no direct evidence that HIV can be transmitted via respiratory secretions.
    c. Universal precautions carry the implicit assumption that any person is potentially infectious.
    d. all of the above
    e. a & c only

[August 1989:734-740]

14. Empyema
    a. is an infection within the pleural space and most commonly occurs as a complication of bronchiectasis.
    b. most commonly forms in nondependent areas of the pleural space.
    c. is—in most cases—a sterile inflammatory exudate.
    d. rarely results in compression atelectasis of the adjacent lung.
    e. a and b

[August 1989:745-747]

15. Measured MIP is likely to be greater at low lung volumes than at high lung volumes because
    a. the diaphragm is in a more advantageous position for contraction.
    b. the stimulus to ventilatory drive may be greater.
    c. breath-holding duration will be longer.
    d. all of the above
    e. a and b only

[September 1989:789-794]

16. In the study by East et al, during severe respiratory depression
    a. spontaneous V_E ceased completely in the PSV + MMV group.
    b. total V_E was less in the PSV + MMV group than in the PSV group.
    c. P_{aCO_2} increased dramatically in the PSV + MMV group.
    d. S_{aO_2} decreased dramatically in the PSV + MMV group.
    e. all of the above

[September 1989:795-800]

17. The "ESP" Infant Cannula for low-flow oxygen delivery
    a. requires less taping for stabilization than the traditional nasal cannula.
    b. has no surface in direct contact with the nasal septum.
    c. is said to facilitate bottle and breast feeding of the infant.
    d. all of the above
    e. b and c only

[September 1989:801-804]

18. In the study by Hess et al, volumes delivered during adult bag-valve ventilation
    a. were unaffected by the number of hands used.
    b. were affected by the size of the hands used.
    c. were affected by the brand of resuscitator used.
    d. all of the above
    e. b and c only

[September 1989:805-810]

19. In the treatment of IRDS, surfactant preparations
    a. do not appear to be of therapeutic value beyond 72 hours after birth.
    b. appear to be most effective when administered prophylactically—immediately upon delivery (preferably before the first breath is taken).
    c. are most effective when instilled after proteinaceous edema fluid has entered the alveoli.
    d. a and c
    e. a and b

[September 1989:811-814]

20. Inspiratory muscle training
    a. may lead to a decrease in the patient's sensation of dyspnea.
    b. is particularly efficacious in the presence of muscle fatigue.
    c. will usually increase muscle strength but not endurance.
    d. will lead to lessening of thoracic hyperinflation.

[October 89:860-867]

21. According to Kacmarek et al, the inclusion of a one-way valve in the system for measuring maximal inspiratory pressure (MIP)
    a. produces higher values than simple airway occlusion only when occlusion time exceeds 15 seconds.
    b. ensures that the lung volume is as low as possible when the measurement is made.
    c. led to the development of arrhythmias and hypotension in many of the study patients.
    d. all of the above
    e. a and b only

[October 89:868-878]

21. According to Kacmarek et al, the inclusion of a one-way valve in the system for measuring maximal inspiratory pressure (MIP)
    a. produces higher values than simple airway occlusion only when occlusion time exceeds 15 seconds.
    b. ensures that the lung volume is as low as possible when the measurement is made.
    c. led to the development of arrhythmias and hypotension in many of the study patients.
    d. all of the above
    e. a and b only

[October 89:868-878]

23. Palatal groove
    a. is a purely congenital anomaly.
    b. may be less serious if the infant is allowed to suck on his orotracheal or endotracheal tube.
    c. usually results from the pressure of the laryngoscope on the alveolar ridge.
    d. may be avoided by the use of a palatal groove appliance.
    e. all of the above

[October 89:879-889]

24. Hypoplasia of tooth enamel
    a. can result from trauma during laryngoscopy and intubation.
    b. can result from 'hereditary' factors such as maternal diabetes.
    c. may be manifested by crack-like lines or flaking of the tooth.
    d. all of the above
    e. a and c only

[October 89:879-889]

25. Interactive videodisc technology is termed *interactive* because
    a. certain scenes in the instructional sequence are animated.
    b. the audiovisual effects are realistic.
    c. the system adjusts instructional sequences to learner input.
    d. the system requires participation by the learner.

[October 89:890-898]

26. Artificial noses
    a. provide a constant resistance to airflow over use time.
    b. show increased resistance as weight of the device increases.
    c. should be replaced at least every 8 hours.
    d. provide decreased resistance with increased airflow.
    e. a and c only

[October 89:902-907]

27. The increase in resistance experienced over time with hygroscopic condenser humidifiers
    a. probably results from the accumulation of moisture in the inserts.
    b. will not affect patients who are on controlled ventilation.
    c. may be important during IMV and T-piece weaning in patients with chronic disease or respiratory muscle dysfunction.
    d. all of the above
    e. a and c only

[October 89:902-907]

28. Which of the following is most correct?
    a. End-tidal P_{CO_2} (P_{etCO_2}) is similar to mean alveolar P_{CO_2} (P_{ACO_2}) during spontaneous breathing.
    b. Tidal volume delivered with positive pressure tends to distribute to gravity-dependent areas of the lung.
    c. End-tidal P_{CO_2} (P_{etCO_2}) is essentially independent of breathing pattern.
    d. In the study of Smith et al, the P_{aCO_2}-to-P_{etCO_2} gradient was significantly lower with SIMV breaths than with spontaneous breaths.
    e. b and c

[November 89:972-975]

29. Which of the following statements is most nearly correct?
    a. Authorities differ little, if at all, in their recommendations for the humidification of inspired gas for intubated patients.
    b. The ANSI standards for heated humidifier water output is 40 mg or 2.2 mol/L of breathing gas.
    c. Heat-moisture exchangers and heat-moisture exchanging filters add less dead space to a ventilator circuit than do large reservoir humidifiers.
    d. Heat-moisture exchangers and heat-moisture-exchanging filters are probably not suitable for patients with large volumes of sputum.
    e. all of the above.

[November 89:976-984]

30. In the Pisut study, the use of an expiratory reservoir in a hand-held nebulizer system for spontaneously breathing patients
    a. increased expiratory resistance.
    b. resulted in the greatest aerosol recovery.
    c. automatically led to enhanced patient response.
    d. enhanced the dissipation of aerosol.
    e. a and d

[November 89:985-988]

31. An expert system
    a. should have the ability to explain its advice and conclusions.
    b. solves problems in a way totally foreign to human methods.
    c. is faster than human subjects because it need not be concerned with the domain expert's experience, judgment, and opinions.
    d. must consist of a knowledge base and a shell and be developed by the use of traditional programming languages.
    e. b and c

[November 89:993-1001]

32. Mang and Obermayer
    a. believe that differences among incentive spirometers in terms of clinical outcome are negligible.
    b. have observed that patients achieve larger inspiratory volumes with devices that impose less work of breathing.
    c. believe that incentive spirometers associated with low imposed work of breathing should be provided to patients vulnerable to inspiratory muscle fatigue.
    d. all of the above
    e. b and c only

[December 89:1122-1128]

33. Hmelo and Axton
    a. concluded that therapists who are involved in performing very complex job tasks are better satisfied than those who must perform many different kind of tasks.
    b. concluded that utilization of high technology increases therapists' job satisfaction.
    c. determined that hemodynamic monitoring, culturing equipment, discharge planning, and physical assessment were all complex tasks.
    d. found that chest physical therapy was the only task that contributed significantly to satisfaction with work.
    e. a and b

[December 1989:1129-1134]

34. In the patient series reported by Adamo et al,
    a. of 120 'complications' noted, only 10 were considered severe.
    b. minor, adverse events did not discourage most patients from continuing with transtracheal oxygen.
    c. previously reported advantages of transtracheal oxygen (increased patient compliance and decreased oxygen flow) were borne out.
    d. potentially serious complications observed included formation of tracheal mucus casts, catheter misplacement, and marked hemoptysis.
    e. all of the above

[February 1990:153-160]

35. LFPPV-ECCO$_2$R
    a. means low-frequency positive-pressure ventilation with extracorporeal CO_2 removal.
    b. accomplishes oxygenation primarily through the use of an extracorporeal membrane lung.
    c. is intended to rest the lung by low peak pressures at high frequency.
    d. all of the above
    e. a and c only

[March 1990:224-231]

36. "Open-loop control" as applied to computerized ventilator management and patient care
    a. threatens the status and job security of the therapist.
    b. is less feasible than closed-loop control because of the limitations of sensors for measuring oxygenation and ventilation.
    c. means that the clinician makes the final decisions related to ventilator management.
    d. all of the above
    e. a and b only

[March 1990:232-240]

37. Which of the following is true?
    a. The use of sucralfate rather than antacids or H2 blockers has led to a significantly lower incidence of lower respiratory tract infections in mechanically ventilated patients.
    b. Klebsiella is the most frequently isolated gram-negative pathogen seen in ARDS.
    c. Ibuprofen appears to have beneficial effects in animals and human beings at risk for ARDS.
    d. all of the above
    e. a and c only

[March 1990:241-246]

38. Which of the following is true?
    a. We know that, without doubt, improved oxygen transport and oxygen consumption leads to improved outcome in critically ill patients.
    b. We know that optimal hematocrit for a critically ill patient is about 30%.
    c. Stroma-free hemoglobin, perfluorocarbons, and encapsulated hemoglobins are examples of blood substitutes.
    d. Complications of red blood-cell transfusion include transfusion reactions, disease transmission, and increased oxygen-hemoglobin affinity.
    e. All of the above

[March 1990:260-272]

39. Functional status
    a. can be defined as the impact of a patient's dyspnea on performance of daily activities.
    b. in the past has been held to be difficult or impossible to measure.
    c. can be validly and reproducibly measured by scales such as the Modified Dysnea Index, the Modified Pneumoconiosis Research Council Index, and the Medical Research Council Index.
    d. all of the above
    e. a and c only

[April 1990:332-341]

40. In his editorial on aerosol delivery to mechanically ventilated patients, Hess concluded from the literature that
    a. More aerosol is deposited in the lung of the intubated patient than in the unintubated patient because no aerosol is deposited in the upper airway.
    b. The closer the MDI is to the threshold of the airway, the more aerosol is deposited in the patient's lung.
    c. Two puffs from an MDI is an adequate dose for 80% of patients studied.
    d. Patient response to bronchodilator by MDI is similar to response by SVN.
    e. a and b only

[May 1990:399-404]

41. In a bench study of home care ventilators, Kacmarek et al found that the imposed work of breathing during spontaneous breathing
    a. increased as inspiratory flowrate increased.
    b. increased with increased resistance to inspiration from the humidifying device.
    c. was decreased in the ventilator systems when compared to work of breathing with an H-valve modification.
    d. all of the above
    e. a and b only

[May 1990:405-414]

42. In the bench study of a bi-level CPAP device, Strumpf et al
    a. found that nose-mask application of the device was inadequate for nocturnal ventilation in their four patients.
    b. found that minute ventilation could be adequately augmented by the device in their four patients.
    c. concluded that the device is probably adequate for all ventilator-dependent home care patients.
    d. concluded that the device adapted well to large variations in airways resistance.
    e. b and d only

[May 1990:415-422]

43. Choose the one most correct answer.
    a. Pressure drop across the capillary tubes or screen mesh of a flow-measuring device is linearly related to flow through the device.
    b. Certain intravenously administered dyes can 'confuse' transcutaneous oxygen monitors.
    c. The Raman spectroscope is probably the foremost application of the Beer-Lambert law.
    d. The phenomenon of paramagnetic susceptibility can be utilized for rapid analysis of CO_2.
    e. all of the above

[June 1990:500-516]

44. Choose the one most correct answer.
    a. The Severinghaus P_{CO_2} electrode is basically a PO_2 electrode that measures CO_2 by difference.
    b. Rapidly changing pressures can be easily measured with a mercury U-tube manometer.
    c. Mass spectrometers are very accurate gas analyzers but are slow because of the ionization process.
    d. CO_2 and water vapor have strong absorption spectra that make them measurable by optical sensors.
    e. a & c only

[June 1990:500-516]

45. Which of the following is true?
    a. Random measurement error is best avoided by careful calibration of measuring devices.
    b. The random error of a particular measurement is the difference between that measurement and the mean of the series of measurements.
    c. If the output of a device is linear, the ratio of output to input is constant.
    d. all of the above
    e. b and c only

[June 1990:520-544]

46. The clinical examination (ie, 'low-tech' monitoring) is less effective than is 'high-tech' monitoring for assessing
    a. ventilatory pattern.
    b. airway function.
    c. clinically important changes in P_{aO_2}.
    d. volume loss.
    e. c and d

[June 1990:546-553]

47. Which of the following is true?
    a. In the normal capnogram the sharp rise in P_{CO_2} that occurs when alveolar gas mixes with dead-space gas is the end-tidal P_{CO_2}.
    b. The rate at which CO_2 is added to the alveolus is determined by alveolar ventilation.
    c. With a normal $\dot{V}/\dot{Q}$, the P_{ACO_2} approximates the P_{aCO_2}.
    d. all of the above
    e. b and c

[June 1990:557-573]

48. Which of the following is true?
    a. Measurement of end-tidal P_{CO_2} is a reliable way of detecting esophageal intubation.
    b. If a victim has been drinking a carbonated beverage, his esophageal P_{CO_2} may remain high for as long as 15 to 20 minutes during manual resuscitator ventilation to the esophagus.
    c. If pulmonary perfusion is low, exhaled CO_2 may be very high because of the resulting high $\dot{V}/\dot{Q}$.
    d. all of the above
    e. b and c only

[June 1990:557-573]

49. The principal advantage of transcutaneous oxygen monitoring over pulse oximetry is
    a. transcutaneous electrodes are less likely to produce a burn on the tender skin of the premature infant.
    b. the transcutaneous monitor is a better detector of hypoxemia.
    c. the transcutaneous monitor automatically tracks P_{CO_2}.
    d. all of the above

[June 1990:577-582]

50. The accuracy of a pulse oximeter reading is not affected by the presence of
    a. a dye such as methylene blue.
    b. a high bilirubin level.
    c. shivering.
    d. low perfusion.
    e. any of the above

[June 1990:584-597]

Calendar of Events

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of month two months preceding the month you wish the ad to run. Submit copy and insertion orders to: Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229.

AARC & AFFILIATES

July 13-15 in Naples, Florida. The AARC presents its annual Summer Forum program at the Registry Resort. Education and management topics are featured. For information, contact the AARC Conventions Department at (214) 243-2272.

July 25-27 in Albuquerque, New Mexico. The NMSRC presents its "Annual Convention and Exhibition 1990" and 3rd Annual Golf Tournament at the Holiday Inn Pyramid. Contact David Southward RRT, program chairman, at (505) 841-1580 or the NMSRC, PO Box 35417, Station D, Albuquerque NM 87176-5417.

August 17 in Dayton, Ohio. The Ohio Society for Respiratory Care presents its 3rd Annual Neonatal-Pediatric Conference, "Options in Ventilation," at the Sinclair Center. Topics include high frequency ventilation, home ventilation, negative pressure ventilation, and the 1991 NBRC Pediatric Specialty Exam. Contact Nancy Telford at (513) 226-8387.

August 23-25 in Savannah, Georgia. The Georgia Society for Respiratory Care presents its Annual Summer Meeting. Special activities include a reception for department managers and a golf tournament. Contact Bob Burnaugh at (404) 944-5189.

September 5-6 in Pittsburgh, Pennsylvania. The Pennsylvania Society for Respiratory Care sponsors its 17th Annual Seminar on Pulmonary Medicine and Physiology at the Sheraton Hotel at Station Square. The conference topics include recent advances in pulmonary medicine. Small discussion groups will focus on specific problems and intervention. Contact Lynda Brynner RRT, Respiratory Care Dept, North Hills Passavant Hospital, 9100 Babcock Blvd, Pittsburgh PA 15237. (412) 367-5428.

September 15-16 in Boise, Idaho. The Idaho Society for Respiratory Care presents its 1990 Annual Educational Seminar at the Anderson Center of St Luke's Regional Medical Center. Contact Jeanne O'Hara, 10 Highway 95, Payette ID 83661. (208) 642-9293.

September 18-19 in Honolulu, Hawaii. The Hawaii Society for Respiratory Care presents its 17th Annual Respiratory Care Conference at the Hilton Hawaiian Village Hotel. Contact Helen M Ono RRT, 1717 Palolo Ave, Honolulu HI 96816. (808) 547-9532.

September 19-21 in St Cloud, Minnesota. The Minnesota Society for Respiratory Care presents its 21st Annual Educational Meeting. Topics include respiratory care practice, current and future. Contact Carolyn Chaon, University of Minnesota Hospitals & Clinic, Cardio-Respiratory Services, Harvard Street at East River Road, Box 247, Minneapolis MN 55455, or phone (612) 625-3976 (8 am-4:30 pm CST).

September 19-22 in Guatemala City, Guatemala. The Central American Association for Respiratory Care presents its 2nd Congress on Respiratory Care at the El Dorado Hotel in Guatemala. Contact Susan P Pilbeam, Respiratory Care, Greenville Tech, PO Box 5616, Greenville, SC 29606-5616. (803) 250-8000, ext 2308.

September 25-27 in Atlantic City, New Jersey. The New Jersey Society for Respiratory Care presents its annual Shore Conference at the Trump Castle Hotel and Casino. Contact Ed Mellon RRT at Shore Memorial Hospital, Somers Point NJ 08244. (609) 653-3729.

September 26-28 in Niagara Falls, New York. The Western New York Chapter of the New York State Society for Respiratory Care hosts the 11th Annual Statewide Respiratory Care Symposium. Neil MacIntyre MD, Bruce Wilson MD, William Ferguson CRTT, and Donald Greenblatt MD are featured speakers. Contact Emilie Walker at Mount St Mary's Hospital, 5300 Military Rd, Lewiston NY, or call (716) 298-2142.

September 26-28 in Charleston, South Carolina. The South Carolina Society for Respiratory Care presents its 19th Annual Meeting, "Challenges of a New Decade," at the Marriott Hotel in Charleston. Contact Sandy Byrdic, SCSRC Annual Meeting, PO Box 8500, Florence SC 29501. (803) 661-3629.

October 3-5 in Lexington, Kentucky. The Kentucky Society for Respiratory Care presents its Annual Meeting, "New Decade—New Direction," at the Radisson Hotel in Lexington. Contact Jim Matchuny at Lexington Community College. (606) 257-1022.

OTHER MEETINGS

October 12-14 in Clearwater Beach, Florida. The American Lung Association of Florida presents a conference on "Respiratory Care for the Pediatric and Neonatal Patient." Contact Richard T Doggett, American Lung Association of Florida, PO Box 8127, Jacksonville FL 32239. (904) 743-2933.

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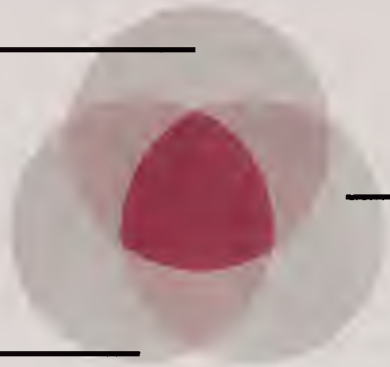

LATERAL ROTATION

Ideal for patients requiring continuous turning for the treatment or prevention of mild to moderate pulmonary complications and other problems that may result from immobility.

The most advanced air-cushion technology with the most advanced critical care frame.

The four-chambered cushion design of the RESTCUE™ Unit, along with its sophisticated microprocessor-controlled airflow system, enables you to select rotation or pulsation or standard low airloss therapies.

"Smart pillows℠" constantly monitor and readjust the rest surface to maintain therapeutic support.

Outer-cushion-chamber firmness helps keep patients centered; helps reduce sliding, shear, and friction during rotation.

HY•TEX™ Breathable Fabric coverlet helps keep patients comfortable and dry.

The **Hill-Rom CENTURY CC® Critical Care** frame of the RESTCUE™ Unit is designed specifically for the critical care environment.

A narrower rest surface, larger wheel casters, and built-in transport convenience make the RESTCUE™ Unit easy to move while also saving space.

A radiolucent window facilitates fluoroscopy; film cassette holder permits radiographic monitoring of line placement and gross changes in lung condition.

STAT•FLAT™ Release Handle simultaneously flattens head of bed and deflates cushions in approximately 10 seconds for rapid CPR.

One-step, easy-grip manual Trendelenburg system quickly angles rest surface.

Built-in full-feature scale weighs only the patient–nothing else.

Until RESTCUE™ Dynamic Air Therapy™, no single unit could meet the changing needs of the critically ill patient.

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RESCUE™

DYNAMIC AIR THERAPY™

The first critical care bed that changes as patient status changes.

"Smart pillows℠" with a unique air-cushion design provide rotation, pulsation, and advanced low airloss modes of therapy and accommodate patients of different size.

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STAT•FLAT™ Release Handle makes unit CPR ready in approximately 10 seconds.

Full-feature scale weighs only the patient—nothing else.

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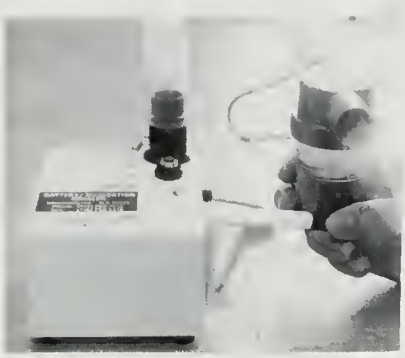
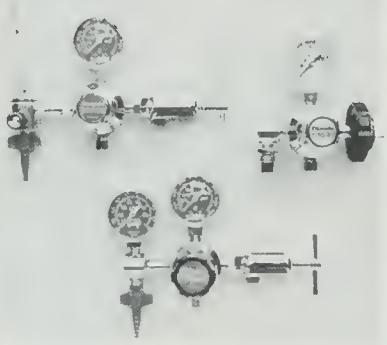
New Products & Services

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE Journal, New Products and Services Dept, 11030 Ables Lane, Dallas TX 75229.

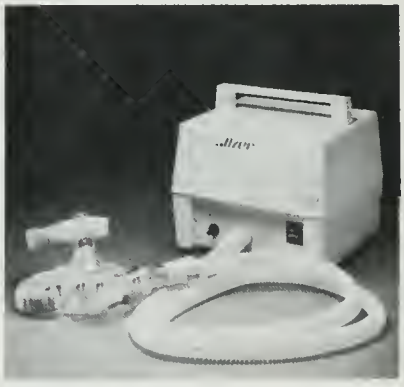


ULTRASONIC NEBULIZER. According to the manufacturer, the Spiral ultrasonic nebulizer is small (measuring 6″ × 6″ × 2″ and weighing only 2.5 lb); mobile (its built-in 12-V battery provides quiet, powerful operation in a car and converts to 110 V for home use); versatile (its 0-10 L/min flow of uniform micro-mist makes it suitable for administrating medication to pediatric and adult patients); and has a sensor that shuts off the unit the instant it detects a lack of fluid in the system. The Spiral carries a 3-year warranty. Medisonic USA Inc, Dept RC, 9600 Main St, Buffalo NY 14031. (716) 759-7214.

CARBON MONOXIDE INDICATOR. The MiniCO Carbon Monoxide Indicator is a noninvasive electronic device that measures the amount of carbon monoxide (CO) present in a sample of exhaled gas. The patient exhales into a sampling balloon attached to the indicator. The result of the oxidation of CO to CO_2 is used to measure the amount of CO present, which is indicated in parts per million (ppm) on a LCD. Using a correlation chart provided with the indicator, an estimate of the %carboxyhemoglobin in the blood can also be made. According to the manufacturer, the MiniCO device is easy to use and the measuring process takes only about a minute to perform; suggested applications include monitoring smoking cessation, health education, and detection of CO poisoning. The MiniCO device comes packaged in a carrying case with operating instructions and a supply of disposable mouthpieces, sampling balloons, and calibration gas. MSA Catalyst Research, Dept RC, 3706 Crondall Lane, Owings Mills MD 21117. (301) 356-2400.



MEDICAL GAS REGULATORS. Ohmeda's new line of Medical Gas Regulators for use with D-, E-, and H-cylinders (oxygen or air) are available in Flowgauge, Flowmeter, and Preset models. The Flowgauge regulators, designed for transport use, can provide 0-15 L/min of gas flow for general patient use or 0-1 L/min for low-flow applications; the Flowmeter regulators are pressure compensated to provide precise delivery of gas in the range of 0-15 L/min; and the Preset regulators are designed to provide uniform pressure of 50 psi from a DISS outlet with a demand valve. According to the manufacturer, each Ohmeda regulator comes equipped with a sintered metal inlet filter that protects the regulator from foreign particles, an encapsulated seat assembly module for ease of maintenance, and a positive-pressure relief valve to protect the regulator against over-pressurization. Ohmeda, Critical Care, Dept RC, 9065 Guilford Rd, Columbia MD 21046-1801. (301) 381-5282.



NASAL CPAP SYSTEM. The ReVitalizer Nasal CPAP System, designed for the treatment of sleep apnea, features a 360° swivel on the hose port, an additional port for supplemental oxygen, and a nonrebreathing valve (in addition to the standard exhalation port located on the nasal mask) designed to prevent the recycling of exhaled air in the event of a power failure. Two Revitalizer models are available: a portable model for travelling and for home use and a clinical model for use during diagnostic sleep studies, featuring remote adjustment capabilities that enable the clinician to evaluate and adjust the pressure (PEEP) without disturbing the sleeping patient. Both models come with a 2-year limited warranty. DeVilbiss Health Care Inc, Dept RC, PO Box 635, Somerset PA 15501-0635. (800) 433-1331 or (814) 443-4881.

RESPIRATORY CARE

Instructions for Authors and Typists

These Instructions are meant to guide authors and typists, including veterans in those roles, in the production of quality manuscripts. Perfection is not expected, but the well-prepared manuscript has the best chance for prompt review and early publication.

General Requirements

Submissions should (1) be related to respiratory care, (2) be planned for one of the publication categories below, and (3) be prepared as indicated in these Instructions. A letter accompanying the manuscript must specify the intended publication category, be signed by all the authors, and, when there are two or more authors, state that "We, the undersigned, have all participated in the work reported, read the accompanying manuscript, and approved its submission for publication."

Publication Categories

Research Article (Study): A report of an original investigation.
Evaluation of a Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification.
Case Report: A report of a clinical case that is uncommon or of exceptional teaching value. The author(s) must have been associated with the case. A case-managing physician must be one of the authors or, if not an author, must supply a letter approving the manuscript.
Case Series: Like a Case Report but including a number of cases.
Review Article: A comprehensive, critical review of the literature and state of the art of a pertinent topic that has been the subject of 40 or more published research papers.
Overview: A critical review of a pertinent topic about which not enough research has been published to merit a Review Article.
Update: A report of subsequent developments in a topic that has been critically reviewed (not necessarily in this journal).
Point of View: A paper expressing the author's personal opinions on a pertinent topic.
Special Article: If a paper does not fit one of the foregoing categories but is pertinent, the editors may consider it as a Special Article.
Editorial: A paper that draws attention to a pertinent concern.
Letter: A signed communication about material published in this journal or on topics of interest or value to readers.
Blood Gas Corner: A brief, instructive case report (real or fictional) involving invasively or noninvasively obtained respiratory care blood data, followed by questions for readers—with answers and discussion.
PFT Corner: Like Blood Gas Corner but involving pulmonary function testing.
Test Your Radiologic Skill: Like Blood Gas Corner and PFT Corner but involving pulmonary-medicine radiography and including one or two 4 × 5 or 5 × 7 inch prints of radiographs. The case must be real.
Review of Book, Film, Tape, or Software: Anyone interested in writing a review can discuss it with an editor.

Editorial Consultation and Author's & Typist's Kit

To discuss a writing project, write to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229 or call 214/243-2272.

Authors are urged to obtain the RESPIRATORY CARE Author's & Typist's Kit. The Kit provides *authors* with specific guidance about writing a research paper, writing a case report, converting to and from SI units, and in-house manuscript review. *Typists* can use the Kit's Model Manuscript, a list of journal name abbreviations, and a copy of these Instructions. The Kit is free from the Journal office.

Preparing the Manuscript

General Concerns—*Typist*

- Double-space ALL lines, including those in references, figure legends, and tables. Do not justify right margins.
- Number pages in upper right corner and leave margins of 1½" or more on all four sides of the page.
- For research articles, follow format of Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
- Meticulously follow instructions for typing references.

General Concerns—*Author:*

- Structure manuscript as specified hereafter.
- Provide all requested information on title page as specified hereafter.
- Proofread manuscript for completeness, clarity, grammar, spelling; be sure all references, figures, and tables are cited in the text.
- Consider having paper reviewed in-house before submission.
- Have all co-authors proofread and approve manuscript and sign submission letter.

Manuscript Structure

Most kinds of papers have standard parts in a standard order. However, papers can vary individually, and not every paper will have all the parts listed here.

Research Article: Title page, abstract page, continuous text (Introduction, Materials & Methods, Results, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends. Please consult "Writing a Research Paper," Respir Care 1985;30:1057 (Dec 1985) and Model Manuscript, Respir Care 1984;29:182 (Feb 1984).
Evaluation of Device/Method/Technique: Title page, abstract page, continuous text (Introduction, Description of Device/Method/Technique, Methods of Evaluation, Results of Evaluation, Discussion), Product Sources page, Acknowledgments page, references, tables, figure legends.
Case Report or Case Series: Title page, abstract page, continuous text (Introduction, Case Summary, Discussion), Acknowledgments page, references, tables, figure legends. Also see "How To Write a Better Case Report," Respir Care 1982;27:29 (Jan 1982).
Review Article: Title page, Table of Contents page, continuous text (Introduction, History, Review of Literature, State of the Art, Discussion, Summary), references. May include figures & tables. No abstract. Table of Contents optional. Other formats may be appropriate.
Overview, Update, Point of View, or Special Article: Title page, text (introduction, message), references, tables, figure legends. No abstract.
Letter: Title page (provide a title), text, writer's name & affiliation, references. Tables & figures may be included. Double-space everything. Write "For Publication" on title page.

Structure: Important Details

Title Page: List title of paper, all authors' full names, degrees, credential letters, professional positions, and affiliations. List correspondence address, telephone number, and reprint address if desired. Name sources of grants or other support. Identify any author's consulting or commercial relationships that pertain to the paper's topic.

Abstract Page: Number this Page 1. List paper's title but omit authors' names. Abstract should be 200 words or less and must be informative, briefly specifying main points of paper, such as methods, results, and conclusions drawn.

Statistical Analysis: In research articles, identify statistical tests and chosen level of significance in the Methods section. In Results section, report actual P values.

Figures (illustrations): All photographs, diagrams, & graphs must be numbered as Figure 1, Figure 2, etc, according to the order in which each is first mentioned in the text. Photographs must be glossy prints 5 × 7 to 8 × 10 inches and should be black & white unless color is essential. Letters and numerals must be neat and large enough to remain legible if figure is reduced in size for publication. Final figures must be of professional quality, but 'rough' sketches may accompany the submitted manuscript, with final figures to be prepared after review. Identify each figure on back with a stick-on label showing figure number and arrow indicating top; omit author's name. Cover label with clear tape so ink will not smudge other prints. Supply three sets of unmounted figures. If figure has been published before, include copyright-holder's written permission to use it.

Figure Legends: List figure legends on a separate page, not on figures. If a figure has been published before, list the source in the legend.

Tables: Type each table on a separate page. Avoid more than 8 columns across. Continue a deep table on following pages. Give each table a number and descriptive title, placed above the table. Double-space ALL lines in tables, including column headings and footnotes.

Drugs: Brand names may be given, but always also show generic names.

Units of Measurement: In addition to conventional units of measure, show SI values and units in brackets after conventional expressions: ie, "PEEP, 10 cm H_2O [0.981 kPa]." For conversion to SI, see RESPIRATORY CARE 1988;33:861-873 (Oct 1988).

Commercial Products: If three or fewer commercial products are named in the text, list the manufacturer's name and location in parentheses the first time each is mentioned. If four or more products are named, do not list manufacturers in the text; instead, name the products and manufacturers in a Products Sources list at the end of the text. Provide model numbers when available.

Abbreviations: Use an abbreviation only if the term occurs several times in the paper. Write out the full term the first time it appears, followed by the abbreviation in parentheses. Thereafter, employ the abbreviation alone. Never use an abbreviation without defining it. Do not create new abbreviations unless absolutely necessary.

References:

- Use references to support statements of fact, indicate sources of information, or guide readers to further pertinent literature.
- Cite only published works—or works accepted for publication. When listing an accepted but still unpublished work, designate the accepting journal's name, followed by "(in press)."
- In the text, cite references by superscript numerals (half space above text), not in parentheses. The first reference cited in the text is number 1, the next is number 2, etc.
- In the reference list, place the cited works in numerical order.
- For the reference list, obtain author names, article and book titles, dates, volume and page numbers from the *original* cited articles and books, not from secondary sources such as other articles' reference lists, which often are inaccurate.
- Type references in medical-journal style. Examples appear at the end of these Instructions. Abbreviate journal names as in *Index Medicus*. A list of many journal-name abbreviations was published in Respir Care 1988;33:1050 (Nov 1988).
- DOUBLE-SPACE the lines of references.
- List ALL authors' names. Do not use "et al" to substitute for names.
- Identify abstracts, editorials, and letters as such. See examples.

Personal Communications, Unpublished Papers, and Unpublished Observations: List unpublished items in parentheses in the text, not in the reference list.

Examples of How To Type References

Notes: Although the examples here are printed with single-spaced lines, please double-space references in manuscripts. Also, note that words in article and book titles are not capitalized—except proper names.

Standard Journal Article:

1. Shepherd KE, Johnson DC. Bronchodilator testing: An analysis of paradoxical responses. Respir Care 1988;33:667-671.

Corporate Author Journal Article:

2. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33:1044-1046.

Article in Journal Supplement:

(Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to allow retrieval.)

3. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, suppl):139s-143s.

Abstract in Journal:

(Abstracts are not strong references; when possible, full papers should be cited. When cited, abstracts should be identified as such.)

4. Lippard DL, Myers TF, Kahn SE. Accuracy of pulse oximetry in severely hypoxic infants (abstract). Respir Care 1988;33:886.

Editorial in Journal:

5. Rochester DF. Does respiratory muscle rest relieve fatigue or incipent fatigue? (editorial). Am Rev Respir Dis 1988;138:516-517.

Letter in Journal:

6. Smith DE, Herd D, Gazzard BG. Reversible bronchoconstriction with nebulised pentamidine (letter). Lancet 1988;2:905.

Personal Author Book:

7. Nunn JF. Applied respiratory physiology. New York: Appleton-Century-Crofts, 1969.

Note: To specify pages cited in a book, place a colon after the year and then list the page(s). Examples: 1969:85 (one page), 1963:85-95 (series of contiguous pages), 1963:85,95 (separated pages).

Corporate Author Book:

8. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Book with Editor, Compiler, or Chairman as 'Author':

9. Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

Chapter in Book:

10. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, eds. Pulmonary medicine. Philadelphia: JB Lippincott, 1977:171-223.

Submitting the Manuscript

After preparing the manuscript according to these Instructions, perform a final proofreading and check for accuracy and completeness. Then mail three copies of the manuscript and three sets of figures to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229 (or Federal Express to RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229). Manuscript copy on IBM-compatible or Macintosh disks in addition to the requisite three hard copies will facilitate processing (Macintosh preferred). Enclose a letter as specified under **General Requirements** at the beginning of these Instructions. Do not submit material that has been published or is being considered elsewhere.

Author's Checklist

1. Is paper for a listed publication category?
2. Does cover letter meet specifications?
3. Is title page complete?
4. Are all pages double-spaced and numbered?
5. Are all references, figures, and tables cited in the text?
6. Are references typed in requested style?
7. Have SI values been provided?
8. Has all arithmetic been checked?
9. Has manuscript been proofread by all authors?

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141	142	143	144	145	146	147	148	149	150	151	152	153	154	155	156	157	158	159	160

I. Type of Instn/Practice
1 ☐ Hosp 500 or more beds
2 ☐ Hosp 300 to 500 beds
3 ☐ Hosp 200 to 300 beds
4 ☐ Hosp 100 to 200 beds
5 ☐ Hosp 100 or less beds
6 ☐ Clinic/Group Practice
7 ☐ Independent RT Provider
8 ☐ Industry (Mfgr/Sales)

II. Department
A ☐ Respiratory Ther
B ☐ Cardiopulmonary
C ☐ Anesthesia Service
D ☐ Emergency Dept

III. Specialty
1 ☐ Clinical Practice
2 ☐ Perinatal Pediatrics
3 ☐ Critical Care
4 ☐ Clinical Research
5 ☐ Pulmonary Func Lab
6 ☐ Home Care/Rehab
7 ☐ Education
8 ☐ Management

IV. Position
A ☐ Dept Head
B ☐ Chief Therapist
C ☐ Supervisor
D ☐ Staff Technician
E ☐ Staff Therapist
F ☐ Educator
G ☐ Medical Director
H ☐ Anesthesiologist
I ☐ Other MD
J ☐ Nurse

V. Are you a member of the AARC?
1 ☐ Yes
2 ☐ No

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1 ☐ Hosp 500 or more beds
2 ☐ Hosp 300 to 500 beds
3 ☐ Hosp 200 to 300 beds
4 ☐ Hosp 100 to 200 beds
5 ☐ Hosp 100 or less beds
6 ☐ Clinic/Group Practice
7 ☐ Independent RT Provider
8 ☐ Industry (Mfgr/Sales)

II. Department
A ☐ Respiratory Ther
B ☐ Cardiopulmonary
C ☐ Anesthesia Service
D ☐ Emergency Dept

III. Specialty
1 ☐ Clinical Practice
2 ☐ Perinatal Pediatrics
3 ☐ Critical Care
4 ☐ Clinical Research
5 ☐ Pulmonary Func Lab
6 ☐ Home Care/Rehab
7 ☐ Education
8 ☐ Management

IV. Position
A ☐ Dept Head
B ☐ Chief Therapist
C ☐ Supervisor
D ☐ Staff Technician
E ☐ Staff Therapist
F ☐ Educator
G ☐ Medical Director
H ☐ Anesthesiologist
I ☐ Other MD
J ☐ Nurse

V. Are you a member of the AARC?
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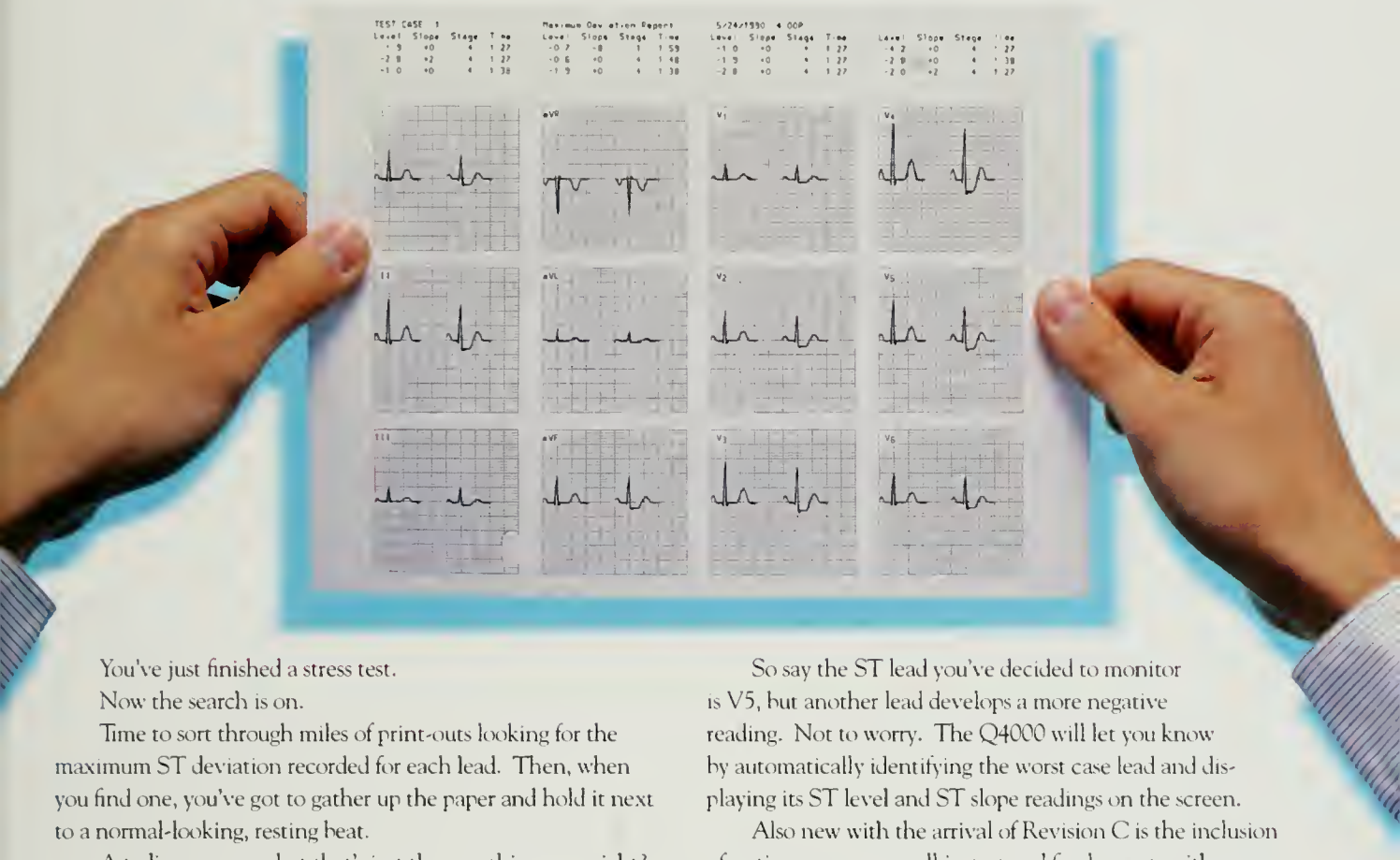

You've just finished a stress test.

Now the search is on.

Time to sort through miles of print-outs looking for the maximum ST deviation recorded for each lead. Then, when you find one, you've got to gather up the paper and hold it next to a normal-looking, resting beat.

A tedious process, but that's just the way things are, right?

Nope. Not any more.

Introducing Revision C Software.

With the Quinton® Q4000 stress test system and the new Revision C software, you get a final report at the end of every test. On it, you'll find a print-out of the maximum ST deviation for each of the 12 leads. Every one printed right next to the resting average waveform for that same lead.

Just like the print-out you see above.

You've got a complete one-page summary. In one convenient, easy-to-compare format.

See More With Revision C.

Revision C also features an ST alert function that watches for the greatest ST depression on all 12 leads.

So say the ST lead you've decided to monitor is V5, but another lead develops a more negative reading. Not to worry. The Q4000 will let you know by automatically identifying the worst case lead and displaying its ST level and ST slope readings on the screen.

Also new with the arrival of Revision C is the inclusion of resting averages on all in-test and final reports with averages. Now they're printed right next to each other, so they're easy to compare.

You've Got Two Calls To Make.

One to us at the number below, to find out more information. The other to your stress test lab (to call off the search).

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For more information or the name of the DHD dealer in your area, contact DHD Customer Service toll-free at: **(800) 847-8000.**

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